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Hydrogen Sulfide Health Effects

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16. ABSTRACT

Health effects literature primarily related to inhalation exposures to hydrogen sulfide was collected, evaluated, tabulated, and summarized. Approximately 350 documents were collected from computerized and manual literature searches covering the period 1887-1981. Pharmacologists and an M.D. epidemiologist rated the documents according to their applicability to the study and their methodology. The 40 documents considered useful for deriving a range of concern for human exposure to hydrogen sulfide from automotive emissions were tabulated. The 60 pages of tables detail the results of acute, repeated dose, and chronic testing of canaries, mice, rats, guinea pigs, chickens, rabbits, cats, monkeys, dogs, pigs, goats, cows, and humans as well as human occupational studies. Most of the documents evaluated are described in an annotated bibliography.

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with Contributions by

Bonnie L. Carson
Cecily M. Beall
Harry V. Ellis III
Larry H. Baker
Joy L. McCann

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For

Emission Control Technology Division
Office of Mobile Source Air Pollution Control
U.S. Environmental Protection Agency
2565 Plymouth Road
Ann Arbor, Michigan 48105

Attn: Robert J. Garbe

PREFACE

This report on health effects of hydrogen sulfide was prepared by Midwest Research Institute (MRI) as Task No. 4 under Contract No. 68-03-2928, "Health Effects Support for the Emission Control Technology Division" for the U.S. Environmental Protection Agency.

Health effects literature primarily related to inhalation exposures to hydrogen sulfide has been collected, evaluated, tabulated, and summarized so that this report can be used to derive a range of concern for human exposure to vehicular atmospheric emissions of hydrogen sulfide.

Task activities were coordinated by the project leader, Mrs. Bonnie L. Carson, Senior Chemist, and task leader, Ms. Cecily M. Beall, Assistant Scientist. Documents were rated and summarized by senior pharmacologist Harry V. Ellis III, of MRI, and epidemiologist Larry H. Baker, M.D., MRI consultant, who is an Associate Professor in the Department of Community Health at the University of Kansas Medical Center. Data were tabulated by Ms. Beall; Ms. Joy L. McCann, Assistant Scientist; and Ms. Eileen M. Horn, Junior Chemist. Ms. Beall and Mrs. Carson contributed to the annotated bibliography. This study was performed under the general supervision of Dr. Edward W. Lawless, Head, Chemical Impact Assessment Section.

Mr. Robert J. Garbe was the project officer for the Emission Control Technology Division, U.S. Environmental Protection Agency, and Ms. Colleen DeMeyer served as Branch Technical Representative.

Approved for:

MIDWEST RESEARCH INSTITUTE

Bruce W Macy
Bruce W. Macy, Director
Center for Technoeconomic
  Analysis

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SUMMARY

This chapter is organized into the following sections: Goals and Methods, Bioassays, Animal Exposures, Human Exposure, International Recommendations and Standards, and Recommended Range of Concern.

GOALS AND METHODS

The purpose of this compilation of data on hydrogen sulfide (H_2S) inhalation exposures is to assist the Emission Control Technology Division (ECTD) of the U.S. Environmental Protection Agency (EPA) to establish the ranges of exposure conditions that are of concern for H_2S in exhausts from vehicles equipped with catalytic converters, and to be able to advise automobile manufacturers thereof. The situations of concern are during malfunctions and during exposures in traffic jams, parking and home garages, and other situations where little dilution of the exhaust is expected before inhalation. Most of the report is, as directed by ECTD, in the form of tables based on the literature reviewed. Data from exposures at levels higher than those of primary concern are included because strictly relevant information was scarce and these related data might prove helpful in assessing health effects at lower levels.

Documents on inhalation effects of H_2S identified from manuals and computerized literature searches were rated in a two-step process by the project pharmacologist and epidemiologist. First, the document received an A, B, C, or D rating according to its applicability for deriving a range of concern for H_2S in automobile emissions. Second, if the paper was not a low-rated, foreign language document,* a theoretical paper, a review, or a nontoxicology experimental paper, it received a numerical score based on itemized features that should be present in an ideal report. For the most part, only A- and B-rated documents were tabulated; but when any C-rated studies involved low-level H_2S exposures, these were also tabulated. Blanks in the tables should be construed as denoting missing information in the documents.

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* Most foreign language articles rated C and D were usually not translated. Each foreign language document tentatively rated A or B from an English language abstract or brief examination of the paper was translated in sufficient degree to judge the experimental design and details. These papers were numerically scored from the translation.

BIOASSAYS

Only five studies using cell cultures or tissue sections were found. They are fully described in Table II-1, and appear to have little information directly useful to this task on human exposure to low H_2S levels.

Tracheal tissue ciliary activity is one protective mechanism against infection. The results of Cralley's (1942) study indicate that this ability is permanently damaged in rabbits by high levels (400-600 ppm) of H_2S. This suggests the possibility of some ciliary inhibition at lower levels. However, no studies were found that used more pertinent H_2S concentrations.

Lung cell phagocytic ability is a second mechanism protecting against infection. The results of Robinson's (1979 and 1980) studies suggest that, in rabbits, the complete expression of whatever cellular lesion that is responsible for a decrease in phagocytic ability following exposure to 50, 60, or 200 ppm H_2S is delayed. The effect appears to be dose-dependent, in terms of both time and concentration. The results of Robinson (1980) indicate that continuous exposure is more detrimental than intermittent exposure (for the same total exposure time), though some of the data from Robinson (1979) might suggest the reverse. Considering the lower levels of H_2S likely to be found in automobile exhaust and the short exposure times to even those levels, extrapolation of these data suggests that there will be little effect of an individual exposure on phagocytic ability. The effect of the repetition of these exposures, however, cannot be estimated. The author plans similar studies at $\leqq$ 50 ppm in the future. The low ratings of these studies are at least partially because only abstracts of the works were available for evaluation.

The study reported in Husain (1976) and Husain and Zaidi (1977) concerns the effect of H_2S (18-500 ppm) on various enzyme activities in rat lungs. A dose-dependent relationship, not necessarily linear, appears to exist, suggesting that some slight changes of unknown significance may occur at lower H_2S levels. The authors believe that these enzyme changes could lead to the deprivation of amino acids needed for protein biosynthesis in lung tissue.

ANIMAL EXPOSURES

This portion of the report summarizes the data available from animal studies. The studies are listed in a series of tables, cited below, and given in Section 3. A summary Table S-1 is given in this section for H_2S levels $\leqq 28.4\ mg/m^3$ (twice the TLV). Unfortunately, most studies involved high and even lethal doses, not minimal ones, so there are little data to aid in determining the lowest range of concern for human exposure.

Studies on acute (< 8 h) exposure to levels $\geqq$ 1,000 mg H_2S/m^3 (up to $\sim$ 46,000 mg/m^3) have been made by four groups of investigators (Lehmann, 1892; Mitchell and Yant, 1925; Weedon et al., 1940; Klentz and Fedde, 1978) on a wide range of animals and the results are summarized as follows: canaries (Table II-1), mice (Table III-2), rats (Table III-5), guinea pigs

TABLE S-1. SUMMARY OF ANIMAL EXPOSURES TO H_2S

Level (mg/m^3)	Time	Species	Effects
28.4	48 h	MUS	Decreased food and water intake and rectal temperature. Weight loss.
28.4	90 d	MUS	Some mortality, survivors showing brain, liver, and lung abcesses. Survivors lost weight, had less endurance, and had instances of bronchopneumonia and hepatitis.
28.4	90 d	RAT	24% mortality, weight loss, lung pathology, and significant changes in many blood parameters.
28.4	1 h/d; 11 d	GPG	Fatigue, somnolence, dizziness, itching, and eye irritation. Decreased lipids in cerebral hemisphere and brain stem.
28.4	90 d	MKY	Weight loss. Changes in many blood parameters.
28.4	21 d	COW	No effect on feed intake, milk production, or heart rate. Slight lacrimation.
22.7	16 h	MUS	Slight restlessness. Normal autopsy.
22.7	16 h	RAT	Slight initial restlessness.
14.2	5 d	MUS	Initial decrease in temperature. Decreased food and water intake, with some recovery by the end.
14.2	41 d	RAT	Also under cold stress, and showed decreased weight gain and food utilization. No changes in blood cells, plasma protein, or weight of liver and lungs.
14.2	4 d	GOAT	Initial decrease in food and water intake.
14	7 h/d; 14 d	DOG	Some coughing. Increased water consumption after each exposure. No change in blood picture or body weight.
12.1	17 d	PIG	NOEL for body weight gain and respiratory system.
10	3 h/d; 3 mo	RAT	Weight gain retarded. Changes in motor chronaxy. Irritation of mucous membranes of trachea and bronchi.
10	6 h/d; 4 mo	RAT	Retarded weight gain. Changes in some blood parameters (increased hemoglobin). O_2 consumption decreased by the end. Changes in lung and heart enzyme activity.
4.86-9.36	6 h/d; 4 mo	RAT	Decreased liver glycogen and lipid levels, blood sugar, and erythrocytes. Increased blood lipids, hemoglobin, and leukocytes.
0.02	12 h/d; 3 mo	RAT	Changes in motor chronaxy. No change in weight. No pathological changes found in sacrificed animals.

(Table III-8), chickens (Table III-10), rabbits (Table III-11), cats (Table III-14), dogs (Table III-18), and goats (Table III-21). At the highest levels there was immediate distress, sometimes death, and usually quick recovery of the survivors. The exposures were longer at the lower levels, symptoms developed more slowly, some deaths still occurred, and recovery frequently was slower. The symptoms imply mucous membrane irritation of the eyes and respiratory tract, and hypoxia. Acute exposures of the same species to $\sim$ 350 to $\sim$ 700 mg H_2S/m^3 caused similar effects in somewhat longer time periods (up to 22 h). The discrepancies between studies on the same species can probably be attributed to the very low numbers of animals used (generally 1 to 4), the different strains used, and variations in individual susceptibility.

In studies of H_2S concentrations < 350 mg/m^3, the symptoms become less severe and the exposure times increase. The acute and intermittent exposures are most directly applicable to this task on human exposure to automobile exhaust in confined or congested situations. The chronic studies, however, can be used to confirm the lack of toxicity or to point out possible adverse effects not considered before.

Hays (1972) found some deaths in mice exposed to even the lowest level studied, 41.6 mg/m^3, with a 16-h exposure (Table III-2). However, 48-h exposure to 28.4 mg/m^3 and 120-h exposure to 14.2 mg/m^3 (Table III-3) produced no deaths, although there were toxic effects (on body weight gain and feed intake). Sandage (1961) exposed mice to 28.4 mg/m^3 for 90 d (Table III-4) and found considerable toxicity: weight loss, increased spontaneous deaths, lung lesions, and an odd pattern of hematologic effects which may be partially compensated anemia (increased erythrocyte production not quite matching increased destruction).

In repeated dose studies, Renne and McDonald (1979 and 1980) found no changes in the major organs of rats exposed to 312.4 mg H_2S/m^3 for 22 d or to 142 mg/m^3 for 7 d (Table III-6). Sandage (1961) found that rats given 28.4 mg/m^3 for 90 d were much like mice: weight loss, increased unscheduled deaths, and lung pathology. Stolpe et al. (1976) found that the combination of 14.2 mg H_2S/m^3 and low temperature (10°C) for 41 d produced additive toxicity (decreased feed utilization, weight gain).

Chronic studies with rats (Table III-7) show definite toxicity (retardation of weight gain, and possible slight nerve cell dysfunction) from repeated exposures (6 h/d for 4 mo) to 10 mg/m^3 (Duan, 1959). One study (Elebekova et al., 1976) reports many changes in hematologic and metabolic parameters after exposure to 4.86 mg/m^3 for 6 h/d for 4 mo.

Most studies (Tables III-8 and III-9) report guinea pigs as responding similarly to rats to H_2S exposure (Mitchell and Yant, 1925; Renne and McDonald, 1979). One study (Renne and McDonald, 1980), however, reports guinea pigs having an inflammatory response after a 22-d exposure to 312.4 mg/m^3, while rats were unaffected.

In repeated dose studies with rabbits (Table III-12), Weise (1933) found little, if any, effects from 20 exposures to 14.2 to 42.6 mg H_2S/m^3. Chronic studies (Table III-13) reported only slight, temporary effects caused by exposure to 28.4 to 35.5 mg/m^3 for 4 h/d for 150 d (Kuwai, 1960) or to 142 mg/m^3 for 30 min/d for 4 mo (Wakatsuki, 1959).

Monkeys may be among the more sensitive species to higher levels of H_2S. Lund and Wieland (1966) found that less than half an hour of 710 mg/m^3 was lethal to rhesus monkeys, with significant individual variability (Table III-16). However, in one of the more useful studies, Sandage (1961) exposed monkeys to 28.4 mg/m^3 for 90 d (Table III-17). He found signficant weight loss but no major specific toxic effects, compared to more severe reactions by mice and rats to the same exposure.

The lowest dose (146.3 mg/m^3) tested by Mitchell and Yant (1925) was lethal to dogs in 16 h (Table III-18). However, Moser (1940) exposed dogs to 130 to 150 mg/m^3 7 h/d for ∿ 8 wk, and found acute signs implying irritation (coughing, thirst, tearing, conjunctivitis). Exposure to 14 mg/m^3 for 7 h/d had no major toxic effects, with complete recovery before the next day's exposure (Table III-19).

Only slight effects were found in larger mammals repeatedly exposed to lower H_2S levels. Curtis et al. (1975) found pigs unaffected by 17 d at 12.1 mg H_2S/m^3 or 19 d at 2.84 mg H_2S/m^3 plus 36 mg NH_3/m^3 (Table III-20). Hays (1972) found several indications of stress in goats exposed to 71 or 142 mg/m^3 for 4 d. He also reported minimal toxic effects (decreased feed and water intake) the first day goats were exposed to 14.2 mg/m^3, but recovery by the end of the 4-d study (Table III-22). Cows were unaffected by 21 d of 28.4 mg H_2S/m^3 (Hays, 1972; Table III-23).

Overall, there are few studies in the area of uncertainty for adverse health effects ($\leqq$ TLV). Most studies involve high and even lethal doses. Specific toxic signs imply that the primary effects of H_2S are irritation of mucous membranes and hypoxia via the well-known subcellular effects. There is limited evidence that large animals are less susceptible and that some tolerance develops. From these animal data, the upper limit of the range of concern should be about 15 mg/m^3, which is the TLV (ACGIH, 1978). There are no animal data for defining a lower limit.

HUMAN EXPOSURE

This section of the report summarizes the data available from human experimental and human environmental (occupational and epidemiological) studies. The most significant studies are cited below, and all studies are described in Sections 4 and 5. Summary Tables S-2 and S-3 are given in this section. A discussion of confounding factors in human environmental studies is included.

Human Experimental Studies

Human studies are relatively limited. Lehmann (1892) did a large number of high dose short-term experiments (see Table IV-1). His basic finding was irritation of mucous membranes (eyes, respiratory tract).

TABLE S-2. SUMMARY OF HUMAN EXPERIMENTAL EXPOSURE TO H_2S

Level (mg/m^3)	Exposure	Table	Effects
3,499-8,165	Acute	IV-1	Eye, nose, and mouth irritations, leading to congestion and secretion. Higher exposures also caused dizziness, trembling, numbness, and heart palpitations. Afterwards, swollen and light-sensitive eyes, headache, fatigue, diarrhea, and bladder tenesmus lasting from several hours to a day.
1,420-4,700	Acute	IV-1	Irritation of eyes, nose, throat, and trachea, leading to tearing, swelling, and catarrh. Symptoms increased with increasing time and concentration. Sometimes irritation and headache continued for several hours after exposure stopped.
994-1,988	Acute	IV-1	Weak irritation of the eyes and throat at the lower levels. At the highest level, bronchitis, rhinitis, and heavy conjunctivitis lasted up to 4 d.
284-568	Acute	IV-1	No signs of irritation, as determined by cursory observation and subjective reaction.
0.20-0.96	Acute	IV-1	All people in the test group detected the odor.
0.08-0.50	Acute	IV-1	Range of odor thresholds within a group.
0.27	Acute	IV-1	Range of odor thresholds within a group.
0.15	Acute	IV-1	Threshold of objectionability (not odor).
0.1	Acute	IV-1	Most people in the test group detected the odor.
0.031-0.09	Acute	IV-1	Some of the people in the test group detected the odor.
0.012-0.03	Acute	IV-1	Range of odor thresholds within a group.

(continued)

TABLE S-2. (concluded)

Level (mg/m^3)	Exposure	Table	Effects
0.012 0.026	Acute	IV-1	Odor was not detected.
0.012-0.013 (2 studies)	Acute	IV-1	Increased light sensitivity-related eye responses.
0.010 (2 studies)	Acute	IV-1	One study found significantly increased light sensitivity-related eye responses. The other study did not.
0.005-0.009	Acute	IV-1	Range of "calculated" odor thresholds within several groups.
0.008	Acute	IV-1	No effect on the ability of the eye to adapt to darkness.
0.00067	Acute	IV-1	Lowest concentration at which all subjects recognized the odor.

TABLE S-3. SUMMARY OF OCCUPATIONAL AND EPIDEMIOLOGICAL EXPOSURES TO H_2S

Level (mg/m^3)	Exposure time	Table	Effects
$\leqq$ 28.4- > 852 (and low concns. of HCN, SO_2, CS_2, hydrocarbons)	"Acute"	V-1	Fatigue, dizziness, unconsciousness with or without respiratory failure. Rapid recovery (0.5 h) except for some nervous symptoms possibly lasting up to 1.5 mo.
326.6	∼ 20 min	V-1	Unconsciousness, cramping, slow and shallow breathing, and low blood pressure. Fully recovered in 6 mo.
40-185	Acute, repeated	V-1	Within several hours, many signs of eye, nose, and throat irriation. Wide variation in individual responses.
∼ 142	-	V-1	Within several hours, many signs of eye irritation. A wide variation in individual response, light cases recovering in a few hours, and severe cases in a week.
13.7-36.6	-	V-1	Eye irritation of varying severity, lasting from several hours to days. Some individuals had repeated episodes.
15-35	"Acute"	V-1	Nausea, weakness, and pain in the chest. Complete recovery within a week, no sequelae.
28.4 (often exceeded)	-	V-1	Fatigue, loss of appetite, irritability, headache, loss of memory, itching, and irritation of the eyes and respiratory tract.
7.1-14.2 (and SO_2 and lower aliphatic compounds)	-	V-1	Respiratory, gastroenteric, eye, and skin irritation.
7.1-14.2 (and SO_2)	5-15 y	V-1	Dermal symptoms suggestive of an allergic-type response. Some lung damage.
< 14.2	-	V-1	Weakness, nausea, dizziness, headache, nervousness, and occasional unconsciousness.

(continued)

TABLE S-3. (concluded)

Level (mg/m^3)	Exposure time	Table	Effects
0-9.94	3 d	V-1	Occasional slight and irregular changes in serum Fe and transferrin levels and in fractions of urinary sulfates.
~ 0.03- ~ 0.43	29 d	V-2	Mild symptoms of nausea, vomiting, headache, shortness of breath, burning eyes, respiratory tract irritation, gastrointestinal complaints, and disturbed sleep.
0.005- 0.300	Chronic	V-2	Headache, weakness, nausea, vision problems, and higher general morbidity rates in those households with $\geqq 0.05$ mg H_2S/m^3.
0.028- 0.055	Chronic	V-2	Babies were poorly developed, underweight, listless, anemic, dyspeptic, and more susceptible to infectious diseases.

Other effects such as headache, fatigue, anorexia, and light sensitivity may have been the result of the stress of the irritation. At his lowest level (284 to 568 mg/m^3 for an hour), there was no apparent irritation.

All other relevant papers involved fractions of a mg/m^3. The greater number involve estimation of odor thresholds. These varied from 0.00067 mg/m^3 (Leonardos et al., 1969) up to 0.27 mg/m^3 (Williams et al., 1977). In addition, Tonzetich and Ng (1976) noted an "objectionability threshold" of 0.15 mg/m^3.

A few experiments have looked for the effects of very low levels of H_2S on sensitivity to light (dark adaptivity). The threshold is about 0.01 mg/m^3 (Baikov, 1963; Duan, 1959).

Human Environmental Studies

These are divided into two groups. Occupational exposures (refinery workers, tunnelers and miners, etc.) are listed in Table V-1. General public exposures are listed as epidemiological studies in Table V-2. In general, these studies are confounded by other noxious exposures and complicated by varying H_2S exposures. Some are also confounded by dubiously matching control groups. Their exposure is greater than a few hours, unlike all the experimental studies above.

The occupational studies (Table V-1) are not useful, except to confirm experimental observations that the major sites for H_2S effects are the eyes and respiratory tract. There are also several occupational studies describing similar H_2S effects, but not giving atmospheric H_2S levels (Demaret and Fialaire, 1974; Aves, 1929; Ahlborg, 1951; Burnett et al., 1977; Chertok, 1968). There is very dubious evidence for a threshold in the range of 1 to 10 mg/m^3 (cf. TLV of 15 mg/m^3). One study of babies chronically exposed to 0.028 to 0.055 mg H_2S/m^3 from their mothers' clothing reported anemia and underdevelopment.

The epidemiological studies (Table V-2) are even less useful. A review of several epidemiological studies in the U.S.S.R. (as well as some experimental data) concluded that chronic exposure to tenths and hundredths of a mg/m^3 can lead to general debility, headaches, vertigo, nausea, and other subjective sensations (Gurinov, 1952).

Confounding Factors

Cigarette smoking is a frequent confounding factor in studies of human exposure to H_2S both because of the respiratory tract effects of the whole smoke and the presence of H_2S in the smoke. Estimates of H_2S levels in cigarette smoke vary with the type of tobacco, type of filter, and the investigator: 81 to 87 μg/nonfilter cigarette and 25 to 89 μg/filter cigarette (Horton and Guerin, 1974), 30 to 49 μg/nonfilter cigarette and 18.7 to 41.5 μg/filter cigarette (Morie, 1971), 3.4 μg/40 mL unfiltered puff and 3.1 μg/40 mL filtered puff (Newsome et al., 1965), and 4.3 μg/puff (nonfilter cigarette; Mattina, 1972). From the latter data it can be estimated that after each puff of a nonfilter cigarette containing 4.3 μg H_2S, the lung will be exposed momentarily to air containing ∿ 6.1 mg H_2S/m^3 (4.3 μg/0.7 L tidal lung volume). This is ∿ 40% of the TLV (15 mg/m^3).

Reviews of human exposure to H_2S frequently include the many studies of employees of the viscose rayon industry. These studies were not included in the tables of this task report because of the confounding presence of CS_2 (which causes symptoms similar to those of H_2S) and H_2SO_4 (which is also toxic). The main symptoms reported are fatigue, nerve effects, and eye irritation (Masure, 1950; Rubin and Arieff, 1945; Kranenberg and Kessener, 1925; Barthelemy, 1939), although other effects have been mentioned (Kriz et al., 1976; Vasil'eva, 1973). Animal (Wakatsuki, 1959; Kuwai, 1960; Masure, 1950; Misiakiewicz et al., 1972) and human (Baikov, 1963) studies indicate at least additive and possibly synergistic effects between H_2S and CS_2 in a mixture. Two of the studies on viscose workers (Barthelemy, 1939; Kranenberg and Kessener, 1925) state that the presence of CS_2 and H_2SO_4 probably plays a part in the formation of eye lesions by increasing sensitivity.

INTERNATIONAL RECOMMENDATIONS AND STANDARDS

Many countries have regulations concerning the levels of H_2S in different situations. Studies have also been done giving recommendations for possible regulations. A summary of such information is given in Table S-4 and in the following paragraphs.

The American Conference of Governmental Industrial Hygienists suggests 14.0 mg H_2S/m^3 as the time-weighted-average threshold limit value and 21 mg/m^3 as the short-term-exposure limit (ACGIH, 1980). The Occupational Safety and Health Administration standard for H_2S exposure is 30 mg/m^3 for a ceiling level. The Maximum Allowable Concentrations (MAC) in air in the U.S.S.R. for 1972 were 10 mg H_2S/m^3 in the workplace, and 0.008 mg/m^3 as both the one-time and average limit in populated places (U.S.S.R., 1972). In 1968, the workplace MAC's for six other countries ranged from 10 to 30 mg H_2S/m^3 (ILO/WHO, 1970).

Several studies have been done by trade or professional associations and, while having no regulatory authority, make recommendations for allowable H_2S concentrations in different situations. The VDI Committee on Air Purification in West Germany recommended atmospheric Permissible Imission Concentrations (PIC) of 0.15 mg/m^3 (half-hour mean value) and 0.3 mg/m^3 (3 x day half-hour mean value). The recommended work-station concentration was 30 mg/m^3 (Verein Deutscher Ingenieure, 1970). In the United States, a study by the Illinois Institute for Environmental Quality (Booras, 1974) recommended an environmental standard for gaseous H_2S of 0.015 mg/m^3 based on 8-h average sample. Two major studies were done in the U.S.S.R. Loginova (1957) recommended 0.03 mg/m^3 for pure H_2S and 0.015 mg/m^3 for H_2S as a constituent of natural gas as the limits in the atmosphere. Gurinov (1952) recommended 0.05 mg/m^3 as the maximum single limit of allowable H_2S in community air and 0.015 mg/m^3 as the maximum average concentration.

TABLE S-4. SUMMARY OF REGULATIONS AND RECOMMENDATIONS FOR HUMAN H_2S EXPOSURE

H_2S level (mg/m^3)	Recommendation/Regulation	Source
30	Recommended work-station concentration	VDI (1970)
21	Short-term-exposure limit in the United States	ACGIH (1980)
14	Time-weighted-average TLV in the United States	ACGIH (1980)
10	Maximum Allowable Concentration in Workplaces in the U.S.S.R	USSR (1972)
0.3	3 x day half-hour mean value, atmospheric PIC	VDI (1970)
0.15	Half-hour mean value, atmospheric PIC	VDI (1970)
0.05	Maximum allowable single limit in community air	Gurinov (1952)
0.005-0.05	Range of concern for automobile emissions	This study
0.03	Limit of pure H_2S in the atmosphere	Loginova (1957)
0.015	Maximum allowable average concentration in community air	Gurinov (1952)
0.015	Atmospheric limit of H_2S as a constituent of natural gas	Loginova (1957)
0.015	Environmental standard based on 8-h average sample	Booras (1974)
0.008	One-time and average limit in populated places in the U.S.S.R	USSR (1972)

RECOMMENDED RANGE OF CONCERN

There is little human evidence for defining a range of concern. The EPA Task 4 Work Directive suggested a preliminary region of uncertainty with regard to the health effects of H_2S of 0.15 to 15 mg/m^3. The TLV is 15 mg/m^3; this seems to be much too high (Booras, 1974). The TLV applies to a self-selected body of healthy workers exposed 8 h/d, 40 h/wk. This is not the situation of concern for automobile emissions.

The human experimental data (Table IV-1) contains a large gap from ∿ 300 mg/m^3 to ∿ 1 mg/m^3, including most of the area of uncertainty. Health effects information is scarce, and most human experimental data concerns odor perception. The best known property of H_2S is its rotten egg odor. The odor threshold could be used as the upper level of the range of concern. However, estimates range from 0.005 to 0.27 mg/m^3 (54-fold) because of individual variability, and the perception of a toxic substance's odor may diminish with the time of exposure so people would cease to be annoyed.

The minimum odor threshold detected was 0.005 mg/m^3 (Adams et al., 1968). The level not affecting eye sensitivity to light was 0.008 to 0.01 mg/m^3 (Duan, 1959; Baikov, 1963). Light sensitivity-related eye responses were seen at 0.012 to 0.013 mg/m^3 (Duan, 1959; Baikov, 1963). Babies were anemic and underdeveloped following chronic exposure to 0.028 to 0.055 mg/m^3 (Glebova, 1950). In an epidemiological study, Loginova (1957) reported nervous and gastrointestinal disturbances during chronic exposure to $\geqq$ 0.05 mg H_2S/m^3.

In conclusion, we suggest 0.005 mg/m^3 as the lower level of the range of concern as a first estimate. Suggested estimates for the upper level of the range of concern are 0.03 mg H_2S/m^3 for babies (a more susceptible group) and 0.05 mg H_2S/m^3 for adults.

SECTION I

INTRODUCTION

This report was compiled as the fourth of several tasks under Contract No. 68-03-2928, "Health Effects Support for the Emission Control Technology Division (U.S. Environmental Protection Agency, Ann Arbor, Michigan)." The goal of the project is to evaluate health effects literature on specific compounds emitted from automobiles equipped with emission-control devices (specifically catalytic converters), not for the purpose of creating a criteria document but to identify a range of concern or a no-observable-effect level for each compound to serve as guidance to automobile manufacturers in their development of future emission-control devices.

The present report was meant to be largely a series of charts or tables of pertinent data with the tests logically ordered according to exposure levels. The narrative summary was not meant to describe again each paper in detail. There are admittedly some disadvantages in not doing so; e.g., some of the gradations in effect that the authors of a particular paper observed may be diluted or lost when the details are spread throughout an exceptionally large table, or between several tables. Papers described in a largely narrative fashion, however, often are difficult to compare. Results that appear within their source paper to be quite definitive may appear less so or even anomalous when juxtaposed in tabular format with other results from similar studies. Hence, the present format was designed to facilitate comparisons.

Literature related to health effects of inhaled H_2S was collected mainly by computer search of TOXLINE and TOXBACK and manual search through the NRC Subcommittee report on hydrogen sulfide. Approximately 350 papers and other documents were evaluated, but only about 40 contained original data suitable for tabulation.

Experimental animal and human exposure studies were evaluated and summarized by a senior Ph.D. pharmacologist. Occupational exposures were rated by an epidemiologist with an M.D. degree. Figure I-1 is the form used for rating documents by the project pharmacologist and epidemiologist. Each document was rated in a two-step procedure according to the applicability of its subject matter and to the quality of the experimental methodology. The letter assigned in rating the document A, B, C, or D was derived from the corresponding lower case letters under item 7 in Figure I-1. Thus, a study was rated A if it directly applies to or assists in establishing a level of concern for exposure to H_2S. The second part of the rating is the

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Article No. 4997-4-_ _ _ _

CHECK WHERE APPROPRIATE:	PAPER DEFECTIVE 0	PAPER IS SUB-STANDARD 1	STANDARD QUALITY 2	SUPERIOR PAPER 3
1. Do they state/limit the problem?				
2. Adequacy of sample				
3. Replicability				
4. Controls/control procedures				
5. Completeness and comprehensibility of results				
6. Validity of conclusions, interpretation of data				

7. Applicability to health effects of H_2S as guidance for establishing a range of concern for H_2S in automobile exhaust.
(circle one)

a. Clearly, directly applies/assists in establishing a range of concern (Chronic human studies; acute exposure of humans if minimal effects.)

b. Research requires major inferences; potentially applicable. (Chronic animal studies; acute human, maximal effect; acute animal, minimal effects.)

c. Useful hints or suggestions; tentatively applicable. (Acute animal, lethal effects; studies in above categories but effects reported not appropriate.)

d. Not directly applicable (peripheral useful information).

Figure I-1 - Form for Report Rating

methodology score. The document reviewer checked off which score should be given for each of the first six items in Figure I-1, and the total was written at the top of the page along with the letter that rated the paper's applicability. In some cases, such as reviews, theoretical papers, and low-rated foreign language documents, a paper may have received an applicability rating (generally C or D) but none on methodology.

Data, including the MRI-assigned rating, from the A-, B-, and some C-rated papers were tabulated by mid-level scientists. Information for each topic heading was carefully sought; so if blanks appear in the table, the reader can generally assume the data were not given. Information which was unclear in the original document but needed for tabulation is preceded in the tables by a qualifying word such as "apparently." Sometimes a group published several papers that described the same tests. To avoid redundancy, all pertinent papers were cited and the test was described as well as possible from all the papers' descriptions.

The final written summary of the tabulated data was also performed by a senior pharmacologist. This summary attempts to reflect objectively the scientific community's thought as a whole and does not reflect the tabular material be weight. The tables reflect the amount of data generated, and the summary puts the evaluated data in perspective with the overall scientific community's opinions.

The references are cited in an annotated bibliography that includes not only each document's rating but also a brief comment on its pertinence (or lack of same) to the study. English titles are given for foreign language documents, and an abbreviation of the language is given in parentheses at the end of the citation.

The report is organized into the following chapters: 2. Bioassay Tests, 3. Experimental Animal Inhalation Exposures, 4. Experimental Human Inhalation Exposures, and 5. Epidemiology. The Summary precedes the entire report and the Annotated Bibliography follows it.

SECTION II

BIOASSAYS

Only five in vitro studies were found in the literature. The results, described in Table II-1, appear to have little information directly useful to this task on determining a range of concern for human exposure to H_2S in automobile exhaust. The data are discussed in the Summary.

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TABLE II-1. BIOASSAYS

Compound and Concentration in mg/m³ (ppm)	Temperature and Humidity	Preparation Exposed	Description of Tests and Duration	Results		Reference and Rating
H_2S 568-1,136 (400-800)	29-31°C, > 95%	Fresh ($\leq$ 2.5 h) excised rabbit tracheal tissue	A 2 x 4 mm section was placed in a tissue chamber, and the irritant gas added at a flow rate similar to that in a living rabbit for 5-10 min. Cilia were observed through a microscope. Time to cessation of activity and then recovery in air or Ringer's solution was determined.	Exposure to 800 ppm for 5 min did not prevent recovery in Ringer's solution. There was cessation of ciliary beating without recovery in air in 5 min at 600 ppm and in 10 min at 400 ppm.		Cralley (1941) D-6
H_2S 712.8 (502)		Rat (albino, ITRC colony) lung homogenate	$\leq$ 5 ml of H_2S gas was injected from a glass syringe into a pre-evacuated glass vial containing 5 ml of the homogenate. The exposed supernatant was kept in the cold for 1 h, then enzyme levels were measured.	Inhibited: Acid phosphatase Alkaline phosphatase GPT (glutamic-pyruvic-transaminase) GOT (glutamic oxalo-acetic-transaminase) ATPase (adenosine triphosphatase) Stimulated: Arginase No effect: Aldolase	 49.0% 43.8% 52.5% 14.0% 41.0% 57.5%	Husain (1976) D-6 Husain and Zaidi (1977) D-11
H_2S 359.3 (253)		Rat (albino, ITRC colony) lung homogenate.	$\leq$ 5 ml of H_2S gas was injected from a glass syringe into a pre-evacuated glass vial containing 5 ml of the homogenate. The exposed supernatant was kept in the cold for 1 h, then enzyme levels were measured.	Inhibited: Acid phosphatase Alkaline phosphatase GPT GOT ATPase Stimulated: arginase No effect: aldolase	 62.0% 34.0% 42.3% 17.2% 37.2% 51.7%	Hasain (1976) D-6 Husain and Zaidi (1977) D-11
H_2S 284 (200)		Alveolar macrophages obtained by lavage of the lungs of New Zealand White rabbits of either sex.	Cell cultures were exposed to H_2S for 48 h in the airstream of a flow-through incubator. Phagocytic ability (as measured by the uptake of polyvinyltoluene beads) and viability (as measured by Trypan Blue and neutral red stains) were immediately determined.	Relative to controls: Phagocytic ability Viability	 2-7% 9-23%	Robinson (1979) C-6

TABLE II-1. (continued)

Compound and Concentration in mg/m³ (ppm)	Temperature and Humidity	Preparation Exposed	Description of Tests and Duration	Results	Reference and Rating
H_2S 284 (200)		Alveolar macrophages obtained by lavage from the lungs of New Zealand White rabbits of either sex.	Cell cultures were exposed to H_2S for 8 h in the airstream of a flow-through incubator. Following 16 h of recovery, there was another 8-h exposure period. Following another 16-h recovery period, there was an additional 8-h exposure. Phagocytic ability was immediately measured.	There was a 32% decrease in phagocytic ability relative to the controls.	Robinson (1979) C-6
H_2S 284 (200)		Alveolar macrophages obtained by lavage from the lungs of New Zealand White rabbits of either sex.	Cell cultures were exposed to H_2S for 8-h in the airstream of a flow-through incubator. After 16 h of recovery, cultures were again exposed for 8 h. Phagocytic ability was determined immediately.	65% decrease in phagocytic ability relative to the controls.	Robinson (1979) C-6
H_2S 284 (200)		Alveolar macrophages obtained by lavage from the lungs of New Zealand White rabbits of either sex.	Cell cultures were exposed to H_2S for 24 h in the airstream of a flow-through incubator. Phagocytic ability and viability were measured immediately and 24 h after recovery.	Immediately after exposure, relative to controls: Phagocytic ability 69-77% Viability 90-95% After 24-h recovery, further decreases in ability were observed relative to controls: Phagocytic ability 7-15% Viability 37-50%	Robinson (1979) C-6
H_2S 153.4 (108)		Rat (albino, ITRC colony) lung homogenate.	$\leq$ 5 ml of H_2S gas was injected from a glass syringe into a pre-evacuated glass vial containing 5 ml of the homogenate. The exposed supernatant was kept in the cold for 1 h, then enzyme levels were measured.	Inhibited: Acid phosphatase 38.5% Alkaline phosphatase 35.0% GPT 38.2% GOT 16.5% ATPase 32.0% Stimulated: arginase 33.0% No effect: aldolase	Husain (1976) D-6 Husain and Zaidi (1977) D-11

TABLE II-1. (continued)

Compound and Concentration in mg/m³ (ppm)	Temperature and Humidity	Preparation Exposed	Description of Tests and Duration	Results	Reference and Rating
H_2S 85.2 (60)		Alveolar macrophages obtained by lavage of the lungs of male New Zealand White rabbits.	Cell cultures were exposed to H_2S for 24 or 48 h in the airstream of a flow-through incubator. Those exposed for 24 h were then given 24-h recovery time. Phagocytic ability was measured at 48 h. Two types of culture methods were used.	Only exposure for 48 h (no recovery time) had an effect on phagocytic ability (~ 20% decrease) of cells cultured on gas-impermeable flasks. Cells cultured on gas-permeable membranes (more similar to in vivo exposure) showed ~ 95% inhibition of phagocytic ability after 24-h exposure with 24-h recovery and after 48-h exposure.	Robinson (1980) C--
H_2S avg 76.5 (avg. 53.9)		Alveolar macrophages obtained by lavage of the lungs of male New Zealand White rabbits.	Cell cultures were exposed to H_2S in the airstream of a flow-through incubator for one of several different regimens: 8 h; 8 h + 16 h delay + 8 h; 8 h + 16 h delay + 8 h + 16 h delay + 8 h; 16 h; or 24 h. Phagocytic ability was measured 24 h after the last exposure (presummably of each regimen).	Exposure for 8 h had no effect on phagocytic ability. Intermittent exposures caused a definite decrease, and continuous exposure caused even greater reductions (0-11% of the controls).	Robinson (1980) C--
H_2S 71 (50)		Alveolar macrophages obtained by lavage from the lungs of New Zealand White rabbits of either sex.	Cell cultures were exposed to H_2S for 20 h in the airstream of a flow-through incubator. After 24-h recovery, phagocytic ability and viability of the cells were determined.	Relative to controls: Phagocytic ability 88% Viability 88-94%	Robinson (1979) C-6
H_2S 56. (45.6		Rat (albino, ITRC colony) lung homogenate.	$\leq$ 5 ml of H_2S gas was injected from a glass syringe into a pre-evacuated glass vial containing 5 ml of the homogenate. The exposed supernatant was kept in the cold for 1 h, then enzyme levels were measured.	Inhibited: Acid phosphatase 33.0% Alkaline phosphatase 19.5% GPT 27.9% GOT 19.5% ATPase 24.0% Stimulated: arginase 23.8% No effect: aldolase	Husain (1976) D-6 Husain and Zaidi (1977) D-11

TABLE II-1. (continued)

Compound and Concentration in mg/m^3 (ppm)	Temperature and Humidity	Preparation Exposed	Description of Tests and Duration	Results		Reference and Rating
H_2S 25.8 (18.2)		Rat (albino, ITRC colony) lung homogenate.	$\leq$ 5 ml of H_2S gas was injected from a glass syringe into a pre-evacuated glass vial containing 5 ml of the homogenate. The exposed supernatant was kept in the cold for 1 h, then enzyme levels were measured.	Inhibited: Acid phosphatase Alkaline phosphatase GPT GOT ATPase Stimulated: arginase No effect: aldolase	 16.8% 11.0% 25.9% 15.9% 13.3% 22.0%	Husain (1976) D-6 Husain and Zaidi (1977) D-11

SECTION III

EXPERIMENTAL ANIMAL INHALATION EXPOSURES

The essential parameters of numerous animal inhalation exposure experiments are tabulated in this section. The primary organization of data is by species, in order of increasing weight (canaries to cows in this case). Within a species, studies are divided by dosing duration: acute exposure ($\leq$ 24 h), repeated exposure, and chronic exposure ($\geq$ 90 d). Within a single table, reported results are listed in order of decreasing exposure level.

The tables have been arranged in the aforesaid manner for the following reasons: (a) there were about 125 separate tests tabulated; (b) there are distinct differences in lung anatomy among the laboratory species used, and the differences seen in their relative responses may have been largely due to these anatomical differences; and (c) by putting the highest concentrations and worst effects first, one can more readily understand the significance of minor or less-severe changes occurring at lower levels. However, a condensation of the data by H_2S concentration is in Table III-24.

In the animal exposure tables in this section, the column headed Total Length of Expt. includes not only the total length of exposure to H_2S but also any recovery time observed in the study. This recovery time was included to note the endurance or reversibility of the toxic effects.

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TABLE III-1. CANARIES--ACUTE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 1,036.6 (730)	Not given	Exposure chamber	Canaries	Not given	None	$\leq$ 2 min, once	-	Falls off perch, unconscious. Dead in 18-20 s. No recovery.	Mitchell and Yant (1925) B-10
H_2S 880.4 (620)	Not given	Exposure chamber	Canaries	2	None	$\leq$ 2 min, once	-	Falls off perch, unconscious 14-20 s. Quick recovery.	Mitchell and Yant (1925) B-10
H_2S 738.4 (520)	Not given	Exposure chamber	Canaries	1	None	$\leq$ 2 min, once	-	Edema and falls off perch, unconsciousness; quick recovery.	Mitchell and Yant (1925) B-10
H_2S 624.8 (440)	Not given	Exposure chamber	Canaries	4	None	$\leq$ 2 min, once	-	Forced respiration, dizziness, edema, 13-20 s unconsciousness. Quick recovery.	Mitchell and Yant (1925) B-10
H_2S 397.6-440.2 (280-310)	Not given	Exposure chamber	Canaries	3	None	$\leq$ 30 min, once	-	Excitement, partial collapse for a short period, then fairly rapid recovery. Edema, forced breathing, unconsciousness, and death. Quick recovery of survivors.	Mitchell and Yant (1925) B-10
H_2S 269.8-298.2 (190-210)	Not given	Exposure chamber	Canaries	4	None	1 h, once	-	In less than 2 min the birds partially collapsed then recovered and were excited. Through the first 0.5 h, there was occasional gasping, edema, forced respiration, dizziness and weakness, and unconsciousness. Some deaths occurred by the end of the hour and the survivors recovered slowly.	Mitchell and Yant (1925) B-10

TABLE III-1. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 198.8 (140)	Not given	Exposure chamber	Canaries	4	None	8 h, once	44 h	Subjects exhibited excitement and one slight case of edema from 30 min-1 h. From 1-4 h forced breathing, dizziness, and weakness were prominent. At 4-8 h very forced breathing, unconsciousness, and death occurred. All the birds that lived through the exposure period died 12-36 h later.	Mitchell and Yant (1925) B-10
H_2S 99.4-142 (70-100)	Not given	Exposure chamber	Canaries	6	None	8 h, once	44 h	Between 30 min and 1 h, one case of slight edema and one case of panting were recorded. At 1-4 h, edema, dizziness, general stupidity, and forced breathing through the mouth were noted. From 4-8 h, very forced respiration, edema, dizziness, unconsciousness, and death occurred. Those surviving the actual exposure died in the following 12-36 h.	Mitchell and Yant (1925) B-10
H_2S 49.7-92.3 (35-65)	Not given	Exposure chamber	Canaries	2	None	18 h, once	-	At 4-8 h, stupid, heavy breathing, edema. At 8-18 h, weakness - inability to perch; death.	Mitchell and Yant (1925) B-10

TABLE III-2. MICE--ACUTE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 1,420 (1,000)	74.6 ± 8.8%, 73.3 ± 3.3°F	Continuous flow inhalation chamber	Mice	4	None	< 1 h, once	< 1 h	50% mortality after 18 min. Animals active during first few minutes. There was marked lacrimation, and all were dead in 20 min. Animals were found to have well-collapsed lungs with small hemorrhages, congested livers and kidneys, and distended stomachs.	Weedon et al. (1940) B-8
H_2S 344 (250)	74.6 ± 8.8%, 73.3 ± 3.3°F	Continuous flow inhalation chamber	Mice	4	None	≤ 8 h, once	8 h	50% mortality after 410 min. At end of 2 h, animals were gasping moderately and their abdomens were distended. All were dead in 8 h. Animals were found to have slightly congested brains, massive hemorrhages of all lung lobes, pale and enlarged livers, moderately distended stomachs with rare minute hemorrhages, and pale kidneys.	Weedon et al. (1940) B-8
H_2S 142 (110)	25°C	Inhalation chamber	Mice, Swiss-Webster	8 F	8 controls treated same, 8 mice deprived of food and water (controls accidentally exposed to 20-30 ppm; see 30 ppm entry)	8 h, once	8 h	Modified lethal concentration duration 50 was 7.5 h. Three mice died and rest were hypothermic. Erythrocyte carbonic anhydrase activity (an enzyme involved in the handling of CO_2, and so reflects the nonoxygen side of respiration) not inhibited. Mice did not eat or drink available food or water.	Hays (1972) B-13

TABLE III-2. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 89.5 (63)	74.6 ± 8.8%, 73.3 ± 3.3°F	Continuous flow inhalation chamber	Mice	4	None	16 h, once	16 h	50% mortality after 804 min. One survived the test but died 23 h later. Animals were found to have congested brains, massive lung hemorrhages, pale and enlarged livers, distended stomachs with few moderate-sized hemorrhages, and pale kidneys.	Weedon et al. (1940) B-8
H_2S 71 (50)	25°C	Inhalation chamber	Mice, Swiss-Webster	8 F	8 controls treated same, 8 deprived of food and water (Controls accidentally exposed to 20-30 ppm; see 30 ppm entry.)	16 h, once	16 h	Modified lethal concentration duration 50 was 15 h. Animals were hypothermic. Erythrocyte carbonic anhydrase activity not inhibited. Food and water intake reduced 70% during exposure.	Hays (1972) B-13
H_2S 41.6 (30)	25°C	Inhalation chamber	Mice, Swiss-Webster	8 F	8 controls treated the same, and 8 mice deprived of food and water (Both groups accidentally exposed to 20-30 ppm H_2S)	24 h, once	13 d	The modified lethal concentration duration 50 was 18.5 h. Three mice died and the rest were hypothermic. Two mice died at 42 h. By day 4 rectal temperatures had increased to pre-exposure level. Food and water intake decreased and mice lost weight. By day 13 weight had returned to normal. Control and fasted groups responded similarly, they lost weight and were hypothermic. At 47 h one of fasted mice died. All had recovered by day 13.	Hays (1972) B-13

TABLE III-2. (continued)

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 22.7 (16)	74.6 ± 8.8% 73.3 ± 3.3°F	Continuous flow inhalation chamber	Mice	4	None	16 h, once	16 h	No abnormal reactions other than an initial slight restlessness which quickly disappeared. Autopsy normal.	Weedon et al. (1940) B-8

TABLE III-3. MICE--REPEATED DOSE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 28.4 (20)	25°C	Inhalation chamber	Mice, Swiss-Webster	7 M	7 controls treated same, 7 mice deprived of food and water	48 h, once	48 h	Food and water intake decreased 20%, rectal temperatures decreased 3.7°C, and animals lost weight. Sleep time as induced by pentobarbital not different from control except deprived group slept longer.	Hays (1972) B-13
H_2S 14.2 (10)	25°C	Inhalation chamber	Mice, Swiss-Webster	6 F	6 F	5 d, once	5 d	No deaths. Body temperature dropped significantly after 48-h exposure but was not significantly different from controls during rest of study. Erythrocyte carbonic anhydrase activity not inhibited. Food and water intake dropped 60% during first 24 h but was inhibited only 28% by day 5 as compared to controls.	Hays (1972) B-13

TABLE III-4. MICE--CHRONIC EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 28.4 (20)	50 ± 10%, 70 ± 5° (Control room up to 86°F)	Exposure chamber	Mice, avg. wt. 25 g	100 M	100 M	90 d, continuous	90 d	Mortality 26% versus 16% in controls. Statistically significant weight loss after 90 d and decrease in swimming endurance compared to controls. Lung pathology present in 33% of animals autopsied compared to 17% in controls. Statistically significant changes in blood parameters at 90-d exposure over pre-exposure levels: leukocytes, hematocrit, hemoglobin, MCV, MCHb, increased; erythrocytes decreased. Compared with controls at 90 d: leukocytes, reticulocytes, hematocrit, hemoglobin, MCV (mean corpuscular volume), MCHb (mean corpuscular hemoglobin), increased; platelets decreased. Mice that died during test period exhibited abcesses of the brain, liver, and lung. Sacrificed animals exhibited instances of bronchopneumonia and hepatitis.	Sandage (1961) B-10

TABLE III-5. RATS--ACUTE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 1,420 (1,000)	74.6 ± 8.8%, 73.3 ± 3.3°F	Continuous flow inhalation chamber	Rats	8	None	< 1 h, once	< 1 h	50% mortality after 14 min. Animals were active during the first 5 min, prostrated in 11 min, and all were dead in 37 min.	Weedon et al. (1940) B-8
								Animals were found to have slightly congested brains, well-collapsed lungs with small hemorrhages, congested livers, distended stomachs, and congested kidneys.	
H_2S 1,122-1,278 (790-900)	Not given	Exposure chamber	Rats	40	Not given	$\leq$ 1 h, once	~ 1 h	0-2 min: unconscious in a few seconds; respiration ceased. A few rats had clonic and tetanic spasms. 2-30 min: respiration stopped. 30 min-1 h: death or rapid recovery after exposure.	Mitchell and Yant (1925) B-10
H_2S 880.4 (620)	Not given	Exposure chamber	Rats	3	Not given	$\leq$ 1 h, once	~ 1 h	0-2 min: great excitement, escape from cages. 2-30 min: stands up in corner, falls over, unconsciousness, clonic spasms. 30 min-1 h: unconsciousness and death or fairly rapid recovery.	Mitchell and Yant (1925) B-10
H_2S 738.4-752.6 (520-530)	Not given	Exposure chamber	Rats	3	Not given	$\leq$ 4 h, once	-	0-2 min: excitement and panting. 2-30 min: forced breathing, eyes closed, weakness, edema. 30 min-1 h: very sick and weak, distressed. 1-4 h: unable to stand, very labored breathing, eyes closed, mouth open, high excitement, and death or slow recovery.	Mitchell and Yant (1925) B-10

TABLE III-5. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 639 (450)	Not given	Exposure chamber	Rats	2	Not given	$\leq$ 4 h, once	-	0-2 min: excitement, evidence of distress. 2-30 min: standing up in corner of cage with nose high, edema. 30 min-1 h: inability to stand, eyes closed, very forced respiration. 1-4 h: semiconsciousness, unconsciousness, great activity, and death or slow recovery.	Mitchell and Yant (1925) B-10
H_2S 440.2-497 (310-350)	Not given	Exposure chamber	Rats	13	Not given	$\leq$ 8 h, once	-	0-2 min: washed face. 2-30 min: washed face, eyes closed, standing up in corner of cage. 30 min-1 h: eyes closed, coughs; forced and rapid respiration; weakness. 1-4 h: very forced respiration; evidence of great distress, spasms, and some deaths. 4-8 h: semiconsciousness, unconsciousness, and death or very slow recovery.	Mitchell and Yant (1925) B-10
H_2S 355 (250)	74.6 ± 8.8% 73.3 ± 3.3°F	Continuous flow inhalation chamber	Rats	8	Not given	22 h, once	22 h	50% mortality not reached. Animals were restless during the first 25 min, then quieted down. First death occurred during the 17 h, 3 were dead at end of 23 h. Animals that died were found to have congested brains, distended and extremely hemorrhagic lungs, distended hearts, congested livers, distended stomachs and intestines, and congested kidneys.	Weedon et al. (1940) B-8

TABLE III-5. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 269.8-340.8 (190-240)	Not given	Exposure chamber	Rats	17	Not given	$\leq$ 18 h, once	-	2-30 min: quiet, standing in corner, head high, washes face. 30 min-1 h: standing up, quiet, in corner 1-4 h: little panting. 4-8 h: forced breathing. 8-18 h: forced breathing and death or very slow recovery after exposure.	Mitchell and Yant (1925) B-10
H_2S 89.5 (63)	74.6 ± 8.8% 73.3 ± 3.3°F	Continuous flow inhalation chamber	Rats	8	None	16 h, once	16 h	50% mortality not reached. Animals unaffected at first but by 16 h they were lethargic and breathing heavily. One rat died, and it had congested brain, half of lung collapsed, moderately dark red liver and kidneys, distended stomach and caecum.	Weedon et al. (1940) B-8
H_2S 22.7 (16)	74.6 ± 8.8% 73.3 ± 3.3°F	Continuous flow inhalation chamber	Rats	8	None	16 h, once	16 h	No abnormal reactions other than a slight initial restlessness.	Weedon et al. (1940) B-8

TABLE III-6. RATS--REPEATED DOSE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 312.4 (220)	Not given	Inhalation	Rats	Not given	Not given	22 d	22 d	No evidence of effect on respiratory tract, kidney, liver, adrenal, heart, gastrointestinal tract, brain, spleen, or eye tissues, examined microscopically.	Renne and McDonald (1980) C--
H_2S 312.4 (220)	Not given	Inhalation	Wistar rats (Charles River Lab.) pregnant females	Not given	Not given	3 h/d; 5 d/wk; For 14 d through gestation, or for 5 d during a portion of major organogenesis	21 d	No evidence of maternal toxicity. Fertility equivalent to the controls. No evidence of embryotoxicity (prenatal mortality or decrease in mean litter weight). Some rib deformities occurred, which were also seen in the controls, although at lower rates.	Andrew et al. (1979) D-9
H_2S 312.4 (220)	Not given	Inhalation	Wistar rats, 14 wk; after exposure, mated to 6-9-wk-old virgin females	10 M	Not given	3 h/d, 7d	~ 13.5 wk	Nondominant lethal effects found in uteri and fetuses of female rats sacrificed 18 d after cohabitation. Fertility (90%), preimplantation loss, avg. no. of corpora lutea, implants, postimplantation loss, and no. of resorptions were all similar to control values.	Andrew et al. (1979) D-9
H_2S 312.4 (220) NH_3 180 (250)	Not given	Inhalation	Rats	Not given	Not given	22 d	22 d	No evidence of exposure effect seen in respiratory tract, kidney, liver, adrenal, heart, gastrointestinal tract, brain, spleen, or eye tissues, examined microscopically.	Renne and McDonald (1980) C--

TABLE III-6. (continued)

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 284 (200)	Not given	Inhalation chamber	Rats	6	6	24 h	48 h	Free cells lavaged from the lungs after 24-h recovery showed increases in the no. of free cells and percent of polymorphonuclear neutrophils (PMN), and decreases in the percent of lymphocytes and macrophages. Changes are indicative of an inflammatory response. Phagocytic ability of the free cells was decreased. Viability was relatively unaffected.	Robinson et al. (1979) C-6
H_2S 142-198.8 (100-140)	Not given	Exposure chamber	Rats	19	Not given	$\leq$ 48 h, once	-	1-4 h: indication of irritation to the nose by continual washing of face. 4-8 h: irritation of nose and eyes. 8-18 h: forced breathing. 18-48 h: death or very slow recovery following exposure.	Mitchell and Yant (1925) B-10
H_2S 142 (100)	Not given	Inhalation	Rats	Not given	Not given	7 d	7 d	No significant histopathologic lesions or clinicopathologic alterations.	Renne and McDonald (1979) C--
H_2S 142 (100) NH_3 180 (250)	Not given	Inhalation	Rats	Not given	Not given	7 d	7 d	No significant histopathologic lesions or clinicopathologic alterations.	Renne and McDonald (1979) C--
H_2S 49.7-92.3 (35-65)	Not given	Exposure chamber	Rats	4	Not given	100 h, once	100 h	1-4 h: showed irritation to nose by continued washing of face. 4-8 h: irritation of nose and eyes. 8-18 h: quiet. 18-48 h: pus in eyes and nose, hair rough. No worse after 100 h continuous exposure.	Mitchell and Yant (1925) B-10

TABLE III-6. (continued)

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 28.4 (20)	50 ± 10%, 75 ± 5°F (Control room up to 86°F)	Inhalation chamber	Rats, Sprague-Dawley, general purpose strain, avg. wt. 175 g	50 M	50 M	90 d, continuous	90 d	Mortality 24% versus 4% in controls. Significant weight loss after 90 d. Lung pathology present in 33% and kidney pathology in 6% of animals autopsied. Statistically significant changes in blood parameters at 90-d exposure over pre-exposure levels: platelets, reticulocytes, hematocrit, hemoglobin, MCV, MCHb, increased. Compared with controls at 90 d: leucocytes and reticulocytes, increased; platelets, decreased.	Sandage (1961) B-10
H_2S 14.2 (10)	50-70% 10°C	Inhalation	Rats	13	15 (2 control groups, one held under optimal temperature, other at 10°C)	41 d	41 d	After 7 wk, significantly less wt. gain, 81% for the test group versus 92% for controls under cold conditions, both compared to controls at an optimal temperature. Food utilization reduced, 132% versus 122% for controls under cold conditions. No differences in blood cells, plasma protein, or wt. of liver and lungs.	Stolpe et al. (1976) B-10
H_2S 14.2 (10) NH_3 71 (50)	50-70% 10°C	Inhalation	Rats	12	20 (2 control groups, one held under optimal temperature, other at 10°C)	50 d	50 d	Significantly less wt. gain, 81% versus 92% for controls under cold conditions. Food utilization reduced, 148% versus 122% for controls under cold conditions. No differences in blood cells, plasma protein, or wt. of liver and lungs.	Stolpe et al. (1976) B-10

TABLE III-6. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 7.1 (5) NH_3 42.6 (30)	50-70% 10°C	Inhalation	Rats	12	13 (2 control groups, one held at optimal temperature, one at 10°C)	Continuous 53 d	53 d	No differences in wt. gain from the cold control, both 92% of rats at optimal temperature. Food utilization reduced, 148% versus 122% for cold conditions. No differences in blood cells, plasma protein, or wt. of liver and lungs.	Stolpe et al. (1976) B-10
H_2S 7.1 (5) NH_3 42.6 (30) Dust 42.34 ± 14.07	50-70% 10°C	Inhalation	Rats	15	15 (2 control groups, one held under optimal temperature, other at 10°C, both exposed to dust same as test rats)	44 d, exposed to dust for 1 h, 2 times/d, 5 d/wk	44 d	Less wt. gain than cold controls, 87% versus 92%. Food utilization reduced, 130% versus 122% for controls under cold conditions. No differences in blood cells, plasma protein, or wt. of liver and lungs.	Stolpe et al. (1976) B-10

TABLE III-7. RATS--CHRONIC EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 50 CO 300	Not given	Inhalation chamber	Rats, albino	Not given	Not given	4 h/d, 6 mo	6 mo	O_2 consumption lowered 17%. Erythrocyte content increased 117%, Hb content not significantly changed. Summation - threshold index down 83% at 2 wk (controls down 93%) but later increased to 120% of original value (controls were 104% of original value). Increased respiration in heart and lung tissue, no change in kidneys. Significant increase in percent of active neutrophils. Increased permeability of vessels.	Mel'nichenko (1968) B-7
H_2S 10	Not given	Inhalation chamber	Rats, albino, 60-90 g	10 M	10 M	12 h/d, "except days off," for 3 mo	17 wk	Wt. retarded compared to controls. Change in the motor chronaxy (a measure of nerve cell function) of rats. In controls chronaxy of straightening is consistently above the chronaxy of flexing. After 2-wk exposure the two reversed and remained that way through 6 wk; over the next 3 wk the two returned to normal positions, reversed again, and finally moved back toward a normal relationship. In the 2 rats killed for study, there was slight irritation of mucous membranes of trachea and bronchi. Changes in appearance of some dendrites in brain cortex.	Duan (1959) B-10

TABLE III-7. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 10 (Concentration reported as MAC, no value given. This value taken from 1972 MAC in U.S.S.R, 1972)	Not given	Inhalation chamber	Rats, albino	~ 26	~ 26	6 h/d	4 mo	Retardation of wt. gain. No significant changes in number of erythrocytes. Hb content of blood significantly increased. Number of leukocytes fluctuated up and down over 4 mo, was higher than controls at end. O_2 consumption increased slightly at beginning, then decreased to 21.4% less than controls. No significant change in succinate dehydrogenase (SDG) activity in lungs or kidneys, 16.7% decrease of SDG activity in heart. Activity of cytochrome oxidase in lungs increased 19.0%.	Aitbaev et al. (1976) B-9
H_2S 9.36 ± 0.51	Not given	Inhalation chamber	Rats, white, 80-200 g	24	14	6 h/d, daily except Sunday, 4 mo	4 mo	Reduced level of glycogen in liver (91.9%), lower blood sugar (69.1%), increased lipids in blood (202.6%), and lower lipids in liver (27.9%). Expressed lipemia present. Lowering of erythrocytes, significant increase of hemoglobin and leukocytes, and slight leukocytosis.	Elebekova et al. (1976) B-11
H_2S 4.86 ± 0.08	Not given	Inhalation chamber	Rats, white, 80-200 g	22	14	6 h/d, daily except Sunday, 4 mo	4 mo	Reduced level of glycogen in liver (68%), lower blood sugar (50.2%), lower lipids in liver (38.8%), and increased lipids in blood (98.6%). Expressed lipemia present. Increase in erythrocytes, hemoglobin, slight increase in eosinophils and segmented neutrophils, slight reticulocytosis, expressed leukopenia, and sharp lowering of monocytes and lymphocytes.	Elebekova et al. (1976) B-11

TABLE III-7. (continued)

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 0.02	Not given	Inhalation chamber	Rats, albino, 60-90 g	10 M	10 M	12 h/d, "except days off," for 3 mo	17 wk	No change in wt. compared to controls. Change in the motor chronaxy of rats. In controls chronaxy of straightening is consistently above the chronaxy of flexing. After 2-wk exposure the two were converging and stayed near the same through 7 wk. During the 7th wk chronaxy of flexing exceeded chronaxy of straightening, then the two moved back toward normal. No pathological changes noted in 2 of the rats killed and examined.	Duan (1959) B-10

TABLE III-8. GUINEA PIGS--ACUTE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 2,130 (1,500)	Not given	Exposure chamber	Guinea pigs	2	Not given	$\leq$ 30 min, once	-	0-2 min: spasms, convulsions, unconsciousness, and cessation of respiration. 2-30 min: death for one, quick recovery for the other.	Mitchell and Yant (1925) B-10
H_2S 1,420-1,562 (1,000-1,100)	Not given	Exposure chamber	Guinea pigs	10	Not given	$\leq$ 30 min, once	-	0-2 min: unconsciousness, spasms, convulsions. 2-30 min: cessation of respiration, spasms, death.	Mitchell and Yant (1925) B-10
H_2S 1,164.4 (820)	Not given	Exposure chamber	Guinea pigs	5	Not given	$\leq$ 30 min, once	-	2-30 min: increased respiration.	Mitchell and Yant (1925) B-10
H_2S 340.8 (240)	Not given	Exposure chamber	Guinea pigs	3	Not given	$\leq$ 18 h, once	-	8-18 h: 2 died, the other had forced respiration and cough with slow recovery following exposure.	Mitchell and Yant (1925) B-10

TABLE III-9. GUINEA PIGS--REPEATED DOSE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 497 (350)	Not given	Exposure chamber	Guinea pigs	3	Not given	$\leq$ 54 h, once	-	4-8 h: forced respiration. 8-18 h: forced respiration, weakness, 2 died, other one very weak and sick. Third guinea pig removed from exposure after 54 h but died 4 d later.	Mitchell and Yant (1925) B-10
H_2S 312.4 (220)	Not given	Inhalation	Guinea pigs	Not given	Not given	22 d	22 d	Mild, acute, suppurative, inflammatory infiltrate in the lungs and trachea. No effect on kidney, liver, adrenal, heart, gastrointestinal tract, brain, spleen, or eye tissues.	Renne and McDonald (1980) C--
H_2S 312.4 (220)	Not given	Inhalation	Guinea pigs, previously exposed to 100 ppm H_2S for 7 d	Not given	Not given	7 d	7 d	Mild increase in the incidence of acute inflammatory lesions of the respiratory tract. 40% incidence of mild interstitial pneumonitis. Mild acute suppurative tracheitis and laryngitis. Mild chronic nephritis in some.	Renne and McDonald (1979) C--
H_2S 312.4 (220) NH_3 180 (250)	Not given	Inhalation	Guinea pigs	Not given	Not given	22 d	22 d	Mild, acute, suppurative, inflammatory infiltrate in the lungs and trachea. Mild, acute, suppurative rhinitis and laryngitis. Slight increase in the incidence and severity of intraluminal calcification of renal cortical tubules. No effect on liver, adrenal, heart, gastrointestinal tract, brain, spleen, or eyes.	Renne and McDonald (1980) C--

TABLE III-9. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 312.4 (220) NH_3 180 (250)	Not given	Inhalation	Guinea pigs, previously exposed to 100 ppm H_2S and 250 ppm NH_3 for 7 d	Not given	Not given	7 d	7 d	Mild increase in the incidence of acute inflammatory lesions of the respiratory tract. 70% incidence of mild interstitial pneumonitis. 40% incidence of mild suppurative bronchiolitis. Mild acute suppurative tracheitis and laryngitis. 100% incidence of mild chronic nephritis.	Renne and McDonald (1979) C--
H_2S 284 (200)	Not given	Inhalation chamber	Guinea pigs	6	6	24 h	48 h	Free cells lavaged from the lungs after 24-h recovery showed increases in the no. of free cells and percent of polymorphonuclear neutrophils (PMN), and decreases in the percent of macrophages and lymphocytes. Changes are indicative of an inflammatory response. Phagocytic ability was slightly decreased. Viability was unaffected.	Robinson et al. (1979) C-6
H_2S 146.3 (103)	Not given	Exposure chamber	Guinea pigs	2	Not given	$\leq$ 48 h, once	-	8-18 h: labored and forced breathing. 18-48 h: death in one case with slow recovery for the other.	Mitchell and Yant (1925) B-10
H_2S 142 (100)	Not given	Inhalation	Guinea pigs	Not given	Not given	7 d	7 d	No significant histopathologic lesions or clinicopathologic alterations.	Renne and McDonald (1979) C--
H_2S 142 (100) NH_3 180 (250)	Not given	Inhalation	Guinea pigs	Not given	Not given	7 d	7 d	No significant histopathologic lesions or clinicopathologic alterations.	Renne and McDonald (1979) C--

TABLE III-9. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 49.7-92.3 (35-65)	Not given	Exposure chamber	Guinea pigs	2	Not given	$\leq$ 100 h, once	-	8-18 h: hair rough. 18-48 h: hair rough, pus in eyes and nostrils, cough. No worse after 100 h continued exposure; all recovered.	Mitchell and Yant (1925) B-10
H_2S 28.4 (20)	40% 30 ± 4°C	Inhalation chamber	Guinea pigs, 400-500 g	6 M	6 M	1 h/d, 11 d	11 d	Fatigue, somnolence, dizziness, itching, and eye irritation were observed. Significant lowering of total lipids and phospholipids in the cerebral hemisphere and brain stem, but no change in the levels in the cerebellum. No change in cholesterol level in any of these regions. Increased lipid peroxidation in the cerebral hemisphere.	Haider et al. (1980) D-11

TABLE III-10. CHICKENS--ACUTE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 5,680	"Ambient temperature and pressure"	Spontaneous breathing through tracheostomy and closed ventilatory system	White Leghorn chickens (Gallus domesticus), Babcock strain, 2.1 kg (anesthetized with sodium phenobarbital to a light plane)	5 M	Each served as its own control; 10 M used for comparison to anesthesia time, and experimental procedures.	$\leq$ 30 min, once	30 min	Exhibited struggling, gasping, apnea, and intermittent bursts of irregular breaths at various times during the exposure. Death within 15 min for all subjects. Cardiac arrest used as death indicator. Variance was significantly different from control group.	Klentz and Fedde (1978) B-12
H_2S 4,260	"Ambient temperature and pressure"	Spontaneous breathing through tracheostomy and closed ventilatory system	White Leghorn chickens (Gallus domesticus), Babcock strain, 2.1 kg (anesthetized with sodium phenobarbital to a light plane)	5 M	Each served as its own control; 10 M used for comparison to anesthesia time, and experimental procedures.	30 min, once	30 min	Exhibition of what appeared to be concentration-related alterations in respiration, with increase in variables within 1 min. Increased respiration continued throughout the first 5 min of exposure then returned to normal during remainder of exposure.	Klentz and Fedde (1978) B-12
H_2S 2,840	"Ambient temperature and pressure"	Spontaneous breathing through tracheostomy and closed ventilatory system	White Leghorn chickens (Gallus domesticus), Babcock strain, 2.1 kg (anesthetized with sodium phenobarbital to a light plane)	10 M	Each served as its own control; 10 M used for comparison to anesthesia time, and experimental procedures.	30 min, once	30 min	Exhibition of what appeared to be concentration-related alterations in respiration, with increase in variables within 1 min. Increased respiration continued throughout the first 5 min of exposure then returned to normal during the remainder of exposure.	Klentz and Fedde (1978) B-12

TABLE III-10. (continued)

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 1,420	Body temperature maintained at 40°C, and ambient pressure	Unidirectional artificial ventilation	Adult White Leghorn chickens, Babcock strain (anesthetized with sodium phenobarbital and secured in dorsal recumbancy)	2 M	Served as own controls	$\leq$ 5 min, once	30 min	Decreased respiratory frequency, increased sternal movements after 30 s. Intrapulmonary CO_2 receptor discharges increased in frequency peaking at 40-80 s. Both birds died within 5 min.	Klentz and Fedde (1978) B-12
H_2S 781	Body temperature maintained at 40°C, and ambient pressure	Unidirectional ventilation	Adult White Leghorn chicken, Babcock strain (anesthetized with sodium phenobarbital and secured in dorsal recumbancy)	1 M	Served as own control	30 min, once	30 min	Decreased respiratory frequency, increased sternal movements after 30 s, sternal movements ceased within 4 min. Intrapulmonary CO_2 receptor discharges increased in frequency.	Klentz and Fedde (1978) B-12
H_2S 738.4	Body temperature maintained at 40°C, and ambient pressure	Unidirectional ventilation	Adult White Leghorn chicken, Babcock strain (anesthetized with sodium phenobarbital and secured in dorsal recumbancy)	1 M	Served as own control	30 min, once	30 min	Decreased respiratory frequency, increased sternal movements after 30 s, sternal movements ceased within 4 min. Intrapulmonary CO_2 receptor discharges increased in frequency, peaking at 40 s.	Klentz and Fedde (1978) B-12

TABLE III-10. (continued)

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 710	"Ambient temperature and pressure"	Spontaneous breathing through tracheostomy and closed ventilatory system	White Leghorn chickens, (*Gallus domesticus*), Babcock strain, 2.1 kg (anesthetized with sodium phenobarbital to a light plane)	10 M	Each served as its own control; 10 M for comparison to anesthesia time, and experimental procedures.	30 min, once	30 min	Respiration not significantly affected. No significant differences in variability comcompared to controls.	Klentz and Fedde (1978) B-12
H_2S 710	Body temperature maintained at 40°C, and ambient pressure	Unidirectional artificial ventilation	Adult White Leghorn chicken, Babcock strain (anesthetized with sodium phenobarbital and secured in dorsal recumbancy)	1	Served as own control	30 min, once	30 min	Increased amplitude of sternal movement, decreased respiratory frequency, respiration ceased after 200 s. Intrapulmonary CO_2 receptor discharges increased in frequency, peaking at 70 s.	Klentz and Fedde (1978) B-12
H_2S 639	Body temperature maintained at 40°C, and ambient pressure	Unidirectional artificial ventilation	Adult White Leghorn chicken, Babcock strain (anesthetized with sodium phenobarbital and secured in dorsal recumbancy)	1 M	Served as own control	30 min, once	30 min	Decreased respiratory frequency, increased sternal movements after 30 s, sternal movements ceased after 10 min. Intrapulmonary CO_2 receptor discharges increased in frequency, peaking at 20 s.	Klentz and Fedde (1978) B-12

TABLE III-10. (continued)

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 497	Body temperature maintained at 40°C, and ambient pressure	Unidirectional ventilation	Adult White Leghorn chicken, Babcock strain (anesthetized with sodium phenobarbital and secured in dorsal recumbancy)	1 M	Served as own control	30 min, once	30 min	Decreased respiratory frequency, increased sternal movements after 30 s, sternal movements ceased within 4 min. Intrapulmonary CO_2 receptor discharges increased in frequency, peaking at 100 s.	Klentz and Fedde (1978) B-12

TABLE III-11. RABBITS--ACUTE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 46,150 (32,500)	Not given	Inhalation chamber	Rabbit, "big and strong"	1, exposed to 1,846 mg H_2S/m^3 for 8 h, 1 wk before	None	2.5 min	12.5 min	Immediate collapse, lying on its side, dyspnea, and body muscle spasms. When exposure stopped, was completely paralyzed, but attempted to walk 3 min later, and was "rather normal" 10 min later.	Lehmann (1892) B-9
H_2S 18,460 (13,000)	Not given	Inhalation	Rabbit, "rather weak"	1	None	3 min	18 min	Staggering, shaky movements, fell on its side, rolled over several times, and stretched rear legs. Strained respiration, rate increasing to 27 from 22 (for 0.5 min). Quick recovery.	Lehmann (1892) B-9
H_2S 11,644 (8,200)	Not given	Inhalation chamber	Rabbit	1, exposed in the previous week to 6,150, 5,112, and 1,846 mg H_2S/m^3 for 2 min, 3.5 h, and 8 h, respectively	None	10 min	"Few hours"	In 2 min, fell on its side, rolled over, nystagmus (rapid, involuntary eye movement, implying CNS problems), and stretching of extremities. After 5 min, began clonic and tonic convulsions of all paws, and series of strong roll-overs. Respiration increased to 42 (for 0.5 min) by 10 min. When removed, could not stand; began crawling in 4 min; recovered fairly fast in the next hours.	Lehmann (1892) B-9
H_2S 5,112-8,236; avg. 6,958 (3,600-5,800; avg. 4,900)	Not given	Inhalation chamber	Rabbit	1	None	1 h at 5,112; 1 h 40 min at 8,236 mg H_2S/m^3	~ 32 h	After 24 h: increasingly restless, then tonic extending movements of the legs, then collapsed on its side, with head twisted; increasing dyspnea; eyes still and half-closed; muscle spasms; mucous membranes red. By the end: spasms in all legs, tonic and tetanic extensions in the neck muscles, clearly nystagmus, dilated pupils, and slightly opaline corneas. 30 h after exposure stopped, head leaning to the right, rolling movements with nystagmus, respiration 30, and would not eat.	Lehmann (1892) B-9

TABLE III-11. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 4,544-6,674; avg. 5,112 (3,200-4,700; avg. 3,600)	Not given	Inhalation chamber	Rabbit, "very big and strong"	1, exposed to 1,300 ppm for 8 h, 2 wk before	None	1 h at 6,674; then 2.5 h at 4,544	3.5 h	Respiration decreased from 63 to 14 (for 0.5 min) after 1.25 h, then increased to 26. No obvious symptoms.	Lehmann (1892) B-9
H_2S 2,982-3,124; avg. ~ 3,124 (2,100-2,200; avg. ~ 2,200)	Not given	Inhalation chamber	Rabbit, "thin"	1	None	1 h 40 min at 2,982; 6 h at 3,124; then 20 min at 2,982 mg H_2S/m^3	8 h	Few symptoms. Respiration decreased in first 4 h, 21-10 (for 0.5 min). After 5 h, increasing dyspnea, 25-40. Sat quietly after exposure stopped.	Lehmann (1892) B-9
H_2S 1,846-2,414; avg. 2,130 (1,300-1,700; avg. 1,500)	Not given	Inhalation chamber	Rabbit	1, exposed for 3 min to 13,000 ppm the day before	None	8 h at 1,846; then 2 h at 2,414 mg H_2S/m^3	10 h	Quiet, lethargic. Respiration increased from 16-12 to 38 (for 0.5 min) in 4 h, then decreased slightly. At 8 h, muzzle slightly opened and strained breathing. By 10 h, occasional coughing.	Lehmann (1892) B-9
H_2S 1,846 (1,300)	Not given	Inhalation chamber	Rabbit, "big and strong"	1	None	8 h	8 h	Quiet. No signs of irritation for the first hour. Respiration 46-41 (for 0.5 min), decreased for next 2.5 h, then increased for 2 h, frequently to 55-65. Strained breathing towards the end. Fine after exposure stopped.	Lehmann (1892) B-9

TABLE III-12. RABBITS--REPEATED DOSE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 1,420 (1,000)		Inhalation chamber	Rabbits	3 M	-	?/d, ?/wk, 18 d	18 d	No obvious disturbances for 3 d. Then increasing irritation of the connective membranes, leading to erosion of the eye membranes. One animal lost weight. Intermittent heavy breathing, but no distinct changes in respiration rate before and after each exposure. No findings on autopsy except slight changes in the stomach or small intestines of 2 animals.	Weise (1933) B-9
H_2S 142 (100)		Inhalation chamber	Rabbits	3 M	None	?/d, ?/wk, 15 d	15 d	Temporarily increased eye secretion. Quiet, moderately exhausted-looking at the end. Slight decrease in respiratory frequency at the end of expt. No weight loss. 2/3 showed, on autopsy and microscopic examination, evidence of increased blood circulation in the gastrointestinal tract (including hyperemia and loss of the villus epithelium in the small intestine).	Weise (1933) B-9
H_2S 50-100 (35.2-70.4)	90%, 20°C	Inhalation chamber	Albino rabbits, 1.5-2.0 kg	Not given	Not given	5 d	5 d	No evidence of any eye lesions was seen.	Masure (1950) B-10
H_2S 14.2-42.6 (10-30)		Inhalation chamber	Rabbits	2 M	None	?/d, ?/wk, 20 d	20 d	No effect on respiration rate, white and red blood cell counts, hemoglobin levels, or appetite. Slight weight decrease. On autopsy, no changes. Some changes in microscopic exam. of the small intestines of one animal.	Weise (1933) B-9

TABLE III-13. RABBITS--CHRONIC EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 142 (100)	Not given	Inhalation chamber	Rabbits, domestic, avg. wt. 20 kg	4	4	30 min/d, daily, 4 mo	4 mo	No measurable abnormal findings in the general condition, body weight, number of erythocytes, serum calcium, total serum protein, and serum protein fraction. There was decline in specific gravity of blood. Some slight changes such as oligochromemia, reticulocytosis, leucopenia, and relative lymphocytosis, which quickly returned to normal after exposure stopped.	Wakatsuki (1959) C-8
H_2S 28.4-35.5 (20-25)	Not given	Inhalation chamber	Rabbits, domestic, avg. wt. 20 kg	5 M	5 M	4 h/d, 150 d	210 d	No change in general state and specific gravity of blood. There was an initial increase in free cholesterol and fall in ester ratio that returned to normal levels after a short period.	Kuwai (1960) B-10

TABLE III-14. CATS--ACUTE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 10,082-11,644; avg. 10,792 (7,100-8,200; avg. 7,600)	Not given	Inhalation chamber	Cat, "young and strong"	1	None	1 h at 11,644; then 49 min at 10,082 mg/m^3	-	For first ~ 1.5 h, alternating restlessness and lying in sleep position, occasional mewing and sneezing, eyes closed, no salivation, and respiration increasing from 13 to 24 (for 0.5 min) then deepening and decreasing to 8. After 100 min, restless, staggering, dilated pupils, urinates and defecates, and increasing dyspnea with respiration at 130 by the end. Some recovery 1 h after exposure stopped; complete recovery "in the following hours."	Lehmann (1892) B-9
H_2S 9,656-10,224; avg. 10,082 (6,800-7,200; avg. 7,100)	Not given	Inhalation chamber	Cats, "young and strong"	2, 1 exposed to 7,600 ppm for ~ 2 h, 3 d before	None	1 h at 9,656; 7 h at 10,224 mg/m^3	-	Previously exposed cat after 3 h: could not stand up, eyes closed with small pupils, continuous secretion of thick saliva, nose dry. Removed after 4 h 16 min: No response to stimulus, irregular respiration, no perceived heartbeat. Slow recovery, fine the next day. The other cat died after 8 h 9 min.	Lehmann (1892) B-9
H_2S 5,112-8,236; avg. 6,958 (3,600-5,800; avg. 4,900)	Not given	Inhalation chamber	Cats	2, 1 exposed in the previous wk to 3,200 and 7,100 and 7,600 ppm for 2-8 h each	None	1 h at 5,112; 1 h 40 min at 8,236 mg/m^3	"a few hours"	Previously exposed cat was immediately in the sleeping position, eyes closed, salivated. Respiration 6-10 (for 0.5 min). The other cat was in the sleeping position after 15 min, respiration 13-23. After 2 h: bloody feces, vomiting, and salivation. Both recovered after several hours.	Lehmann (1892) B-9
H_2S 7,952 (5,600)	Not given	"Box"	Cat, ~ 3 kg	1	None	41 min	41 min	Quiet and slightly sleepy. No other symptoms.	Lehmann (1892) B-9

TABLE III-14. (continued)

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 4,544-6,674; avg. 5,112 (3,200-4,700; avg. 3,600)	Not given	Inhalation chamber	Cat	1, exposed to 1,400 ppm for 10 h, 1 wk before, and 1,300 ppm for 8 h, 2 wk before	None	1 h at 6,674; 2.5 h at 4,544 mg/m^3	3.5 h	Immediately lay on its stomach. Respiration decreased from 16 to 8 (0.5 min). Eyes closed, half asleep. Weak continuous secretion of thick saliva after 12 min.	Lehmann (1892) B-9
H_2S 5,396 (3,800)	Not given	"Box"	Cat, 3 kg	1	None	65 min	65 min	No symptoms.	Lehmann (1892) B-9
H_2S 2,982-3,124; avg. ~ 3,124 (2,100-2,200; avg. ~ 2,200)	Not given	Inhalation chamber	Cats, "young, strong"	2, 1 exposed to 7,100-7,600 ppm for 10 h, 3 or 6 d before	None	1 h 40 min at 2,982; 6 h at 3,124; 0.5 h at 2,982 mg/m^3	8 h	Previously exposed cat lay on its side, respiration ~ 14 (for 0.5 min), strong pulse (51). At end, was half narcotized, had weak reaction to stimulus, could barely walk, occasional salivation, and had red ears. Other animal: no symptoms for 3 h, then lay on its side.	Lehmann (1892) B-9
H_2S 1,846-2,414; avg. 2,130 (1,300-1,700; avg. 1,500)	Not given	Inhalation chamber	Cat	1	None	8 h at 1,846; 2 h at 2,414 mg/m^3	10 h	No clear signs of illness. Quiet, lying on floor. Respiration 10-17 (for 0.5 min). Towards the end, nose slightly wet, occasional coughing. Fine after exposure stopped.	Lehmann (1892) B-9
H_2S 1,846 (1,300)	Not given	Inhalation chamber	Cat	1, with 10-h exposure to 1,500 ppm, 2 d before	None	8 h	8 h	Quiet, lying down. Respiration 8-12 (for 0.5 min), changing after 2 h. Salivary secretions for ~ 4.5 h. Recovered quickly.	Lehmann (1892) B-9

TABLE III-15. CATS--REPEATED DOSE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 1,420 (1,000)	Not given	Inhalation chamber	Cats	2	None	12 h/d, ?/wk, 18 d	18 d	Moderate hoarseness the only sign of illness. No changes in the gastrointestinal tract except increased mucous membrane secretion in one cat. The other cat had severe edema of the lung.	Weise (1933) B-9

TABLE III-16. MONKEYS--ACUTE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 710 (500)	Not given	Inhalation chamber	Rhesus monkey	1	None	35 min, once	< 1 h	Rubbing of eyes, intense gasping decreasing in intensity until heart and respiration stopped. Died after 35 min, blood sugar level at time of death 640 mg%. No discernible pathological changes in any area of brain, nor in kidneys or heart.	Lund and Weiland (1966) B-8
H_2S 710 (500)	Not given	Inhalation chamber	Rhesus monkey	1	None	25 min, 3 d later another 17-min exposure	8 d	Frequent rubbing of eyes, gasping, and sudden unconsciousness. After 25 min respiration stopped, animal revived and was exposed again on 3rd d. In second exposure animal became unconscious after 17 min and died 5 d later. Blood sugar was 200 mg% day of death. Pathological changes in main ganglia of brain characterized by spot-like cellular reactions and vascular changes. Extensive changes in cerebral cortex including cellular and vascular changes.	Lund and Wieland (1966) B-8
H_2S 710 (500)	Not given	Inhalation chamber	Rhesus monkey	1	None	22 min, once	10 d	Frequent rubbing of eyes, gasping, and sudden unconsciousness after 22 min. Exposure ended, it regained consciousness after 140 min, but was somnolent, moved little, was disoriented, and had little appetite. It improved only slightly before being killed at 10 d. Pathological changes in brain, very extended cortex losses in the middle cortex strata in the region of the parietal brain and occipital brain. No visible pathological changes in main ganglia of brain, kidney, heart, and liver.	Lund and Wieland (1966) B-8

TABLE III-17. MONKEYS--CHRONIC EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Ratin
H_2S 28.4 (20)	50 ± 10%, 57 ± 5° (Control room up to 86°F)	Inhalation chamber	Rhesus monkeys, avg. wt. 1.7 kg	10 M	10 M	90 d	90 d	No deaths. Statistically significant weight loss after 90 d. Statistically significant changes in blood parameters at 90 d exposure over pre-exposure levels: erythrocytes and amylase increased, and MCHb and MCHbC (mean corpuscular hemoglobin concentration) decreased. Compared with controls at 90 d: MCHbC decreased, and urine alkaline phosphatase increased.	Sandage (1961) B-10

TABLE III-18. DOGS--ACUTE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 48,280 (34,000)	Not given	"Box"	Dog, ~ 6.6 kg	1	None	~ 2 min	~ 4.5 h	Alternating clonic and tonic convulsions, then moribund but with strong heartbeat. Afterwards, needed artifical "reanimation." 8 min later, deep respiration, cramps, attempted to get up, urinated and defecated. By 4.5 h, passably recovered - eating, and no inflammation symptoms of mucous membranes.	Lehmann (1892) B-9
H_2S 7,952 (5,600)	Not given	"Box"	Dog, ~ 6.6 kg	1	None	41 min	-	After 2 min, was moribund, with stretching spasms, dilated pupils, and slow, very deep respiration. Some recovery, but with epileptic attacks interrupted by euphoric periods. After 20 min of recovery, was able to walk waveringly. The next day had injected conjunctiva and walked stiffly. Eventually recovered.	Lehmann (1892) B-9
H_2S 5,396 (3,800)	Not given	"Box"	Dog, 6.6 kg	1	None	65 min	65 min	Restless, sneezing, blinking, slightly twitching legs, and yawning in the first 10 min. By 15 min, slight gasping and choking, with pounding carotids. Then itching nose, "flabby standing," and "decreased intelligence." Improving by 30 min, and no disturbance at the end.	Lehmann (1892) B-9
H_2S 2,130-2,272 (1,500-1,600)	Not given	Exposure chamber	Dogs	9	Not given	$\leq$ 30 min, once	-	0-2 min: spasms, respiration stops. 2-30 min: death.	Mitchell and Yant (1925) B-10
H_2S 1,817.6 (1,280)	Not given	Exposure chamber	Dogs	4	Not given	$\leq$ 30 min, once	-	0-2 min: spasms, respiration stops. 2-30 min: death.	Mitchell and Yant (1925) B-10

TABLE III-18. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 1,420-1,618.8 (1,000-1,140)	Not given	Exposure chamber	Dogs	8	Not given	$\leq$ 30 min, once	-	0-2 min: falls over unconscious, spasms, convulsions, respiration stops. 2-30 min: death.	Mitchell and Yant (1925) B-10
H_2S 1,207-1,263.8 (850-890)	Not given	Exposure chamber	Dogs	3	Not given	$\leq$ 30 min, once	-	0-2 min: falls over unconscious, spasms, convulsions. 2-30 min: respiration stops, death.	Mitchell and Yant (1925) B-10
H_2S 1,079.2-1,136 (760-800)	Not given	Exposure chamber	Dogs	2	Not given	$\leq$ 1 h, once	-	0-2 min: falls over unconscious; spasms, convulsions. 2-30 min: respiration stops, spasms. 30 min-1 h: respiration stopped and 1 animal died; other taken out and recovered.	Mitchell and Yant (1925) B-10
H_2S 497 (350)	Not given	Exposure chamber	Dogs	2	Not given	$\leq$ 16 h, once	-	2-30 min: lacrimation and nervousness. 30 min-1 h: depression. 1-4 h: depression to stupor. 4-8 h: labored breathing, hemorrhage, and death.	Mitchell and Yant (1925) B-10
H_2S 340.8 (240)	Not given	Exposure chamber	Dogs	2	Not given	$\leq$ 16 h, once	-	1-4 h: depression. 4-8 h: forced respiration, lacrimation. 8-16 h: both died.	Mitchell and Yant (1925) B-10
H_2S 146.3 (103)	Not given	Exposure chamber	Dogs	2	Not given	$\leq$ 16 h, once	-	4-8 h: lacrimation and depression. 8-16 h: pus in eyes, death intense.	Mitchell and Yant (1925) B-10

TABLE III-19. DOGS--REPEATED DOSE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 130-150	Not given	Inhalation chamber	Dogs	4 (2 exposed to 14 mg H_2S/m^3 for 2 wk prior to this exposure)	Apparently none	7 h/d, ~ 8 wk	~ 8 wk	(No effect on 2 dogs during 2 wk at lower exposure, except increased drinking.) At higher concentration, dogs were less lively, nauseous, ate less, and drank more. All dogs coughed and had increased secretion of tear ducts and one developed moderately strong conjunctival infection. Nausea and breathing difficulties disappeared shortly after removal from exposure chamber each day. No significant blood changes.	Moser (1940) B-11
H_2S 14	Not given	Inhalation chamber	Dogs	2	Apparently none	7 h/d, 14 d	14 d	Occasional coughing. Increased liquid consumption and reluctance to eat shortly after each poisoning. Very lively on returning to chamber the next day, with no signs of poisoning. No change in blood picture or body weight.	Moser (1940) B-11

TABLE III-20. PIGS--REPEATED DOSE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 12.1 (8.5)	17-19°C	Inhalation chamber	Pigs, crossbred, 13.2 kg	3 (All of the same sex, unspecified)	3	17 d	-	No effect on body wt. gain. No pathological changes in respiratory system.	Curtis et al. (1975) C-10
H_2S 2.84 (2) NH_3 36 (50)	17-19°C	Inhalation chamber	Pigs, crossbred, 13.2 kg	3 (All of the same sex, unspecified)	3	19 d	-	No effect on body wt. gain. No pathological changes in respiratory tract.	Curtis et al. (1975) C-10

TABLE III-21. GOATS--ACUTE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 1,817.6-1,888.6 (1,280-1,330)	Not given	Exposure chamber	Goats	4	Not given	$\leq$ 30 min, once	-	0-2 min: falls over unconscious, spasms, and cessation of respiration. 2-30 min: death.	Mitchell and Yant (1925) B-10
H_2S 1,420-1,562 (1,000-1,100)	Not given	Exposure chamber	Goats	4	Not given	$\leq$ 30 min, once	-	0-2 min: distress and excitement, bleats. 2-30 min: falls over unconscious, spasms, convulsions, and cessation of respiration, death.	Mitchell and Yant (1925) B-10
H_2S 1,164.4 (820)	Not given	Exposure chamber	Goat	1	Not given	$\leq$ 30 min, once	-	2-30 min: increased respiration.	Mitchell and Yant (1925) B-10

TABLE III-22. GOATS--REPEATED DOSE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 142 (100)	17°C	Inhalation chamber	Goats, mixed breed, 3-4 y, 35-75 kg	6 F	Served as own controls	4 d, once	4 d	Respiration frequency decreased, rectal temperature increased 0.7°C, returning to near normal after 4 d. Food and water intake decreased 37 and 25%, respectively, during first 2 d, regaining near normal levels after 4 d. Plasma cortisol increased but returned to normal levels after 4 d of exposure.	Hays (1972) B-13
H_2S 71 (50)	17°C	Inhalation chamber	Goats, mixed breed, 3-4 y, 35-75 kg	4 F	Served as own controls	4 d, once	4 d	Plasma cortisol increased (possibly indicating stress) and rectal temperature increased 0.7°C, but both returned to near normal after 4 d of exposure. Food and water intake decreased by 64% during the 2nd d, but increased some after 4 d.	Hays (1972) B-13
H_2S 14.2 (10)	17°C	Inhalation chamber	Goats, mixed breed, 3-4 y, 35-75 kg	4 F	Served as own controls	4 d, once	4 d	Food and water intake decreased by 20 and 63% respectively on first day, but returned to near normal levels thereafter.	Hays (1972) B-13

TABLE III-23. COWS--REPEATED DOSE EXPERIMENTAL EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m³ (ppm)	Humidity and Temperature	Mode of Exposure	Species/ Strain/ Age/Weight	No. of Test Animals	No. of Controls	Duration and Frequency of Exposure	Total Length of Expt.	Effects	Reference and Rating
H_2S 28.4 (20)	50%, 20°C	Inhalation through exposure hood over cow's head	Cows (lactating), Holstein, avg. wt. 499 kg	3	Served as own controls prior to exposure	21 d, once	71 d	No effect on feed intake, milk production, or heart rate. Slight lacrimation.	Hays (1972) B-13

TABLE III-24. SUMMARY OF ANIMAL EXPOSURES TO H_2S

Level (mg/m^3)	Time	Species	Effects
7,952-48,280	2-40 min	DOG	Cramping, spasms, convulsions, slow and deep respiration, epileptic-type attacks. Very slow recovery.
5,112-46,150	2 min-~ 3 h	RBT	Weakness, staggering, collapse, spasms, dyspnea, mucous membrane irritation. Fairly rapid recovery.
5,112-11,644	~ 2 h-~ 8 h	CAT	Restlessness, weakness, eye and nose irritation, changes in respiration. One died, the rest recovered in several hours.
1,846-7,952	40 min-10 h	CAT	Few symptoms: quiet, slight respiration changes, occasional coughing, salivary secretions. Fine after exposure. One previously exposed cat had more severe reactions.
1,846-6,674	2-10 h	RBT	Increased respiration, lethargy, occasional coughing. Fine after exposure stopped.
5,680	30 min	CKN	Gasping, apnea, cardiac arrest, and death.
5,396	65 min	DOG	Signs of irritation, gasping and choking, and weakness for 30 min. Then apparent habituation (Lehmann, 1892).
2,840-4,260	30 min	CKN	Increased respiration for 5 min, then normal.
1,079-2,272	30 min-1 h	DOG	Spasms, convulsions, unconsciousness, respiration stops, then death (Mitchell and Yant, 1925).
1,420-2,130	30 min	GPG	Spasms, convulsions, unconsciousness, and death.

TABLE III-24. (continued)

Level (mg/m^3)	Time	Species	Effects
1,888.6	30 min	GOAT	Unconsciousness, spasms, convulsions, respiration stops, and death.
1,420	18 d	RBT	Increased irritation of mucous membranes, intermittent heavy breathing, slight intestinal changes found on autopsy.
	12 h/d; 18 d	CAT	One had increased mucous membrane secretions, and one had severe edema.
1,420	20 min	MUS	Marked lacrimation, all died. Lung, liver, kidney, and stomach damage.
440-1,420	1-8 h	RAT	Excitement, forced breathing, weakness, spasms, unconsciousness, and sometimes death. Survivors recovered more quickly after short, high exposures than after longer, low exposures. Damage to all major organs.
1,164.4	30 min	GPG	Increased respiration.
	30 min	GOAT	Increased respiration.
6,248-1,036.6	2 min	CAN	Edema, dizziness, unconsciousness. Quick recovery.
710	30 min	CKN	No effect on respiration.
710	22-35 min	MKY	Eye irritation, gasping, unconsciousness, and death. Pathological changes in the brain, but not in the kidney, heart, or liver.

TABLE III-24. (continued)

Level (mg/m^3)	Time	Species	Effects
340.8- 497	54 h	GPG	Forced breathing, weakness, immediate or delayed death in some, slow recovery of survivors.
146.3- 497	16 h	DOG	Lacrimation, nervousness, forced breathing, death.
49.7- 440.2	30 min- 18 h	CAN	At higher levels, immediate partial collapse followed by some recovery. Then for all levels: gasping, forced breathing, edema, weakness, unconsciousness, and immediate or delayed death.
269.8- 355	16-22 h	RAT	Quiet, forced breathing, signs of irritation, < 50% mortality, and slow recovery of survivors. Damage to lungs, heart, liver, stomach, kidneys, and intestines.
312.4	3 h/d; 5-14 d	RAT	No dominant lethal effects induced during spermatogenesis in male rats. No embryotoxicity following exposure of pregnant females.
312.4	22 d	RAT	NOEL for major organs.
312.4	14 d and 22 d	GPG	Mild acute inflammatory lesions of the respiratory tract. NOEL for other major organs.
284	24 h	RAT GPG	Changes in lavaged lung cell composition indicating inflammatory response.
130- 150	7 h/d; 8 wk	DOG	Quiet, nausea, coughing, moderate conjunctival irritation, breathing difficulties at the end of each day. No significant blood changes.
146.3	48 h	GPG	Forced breathing, some death, slow recovery of survivors.
142	?/d; 15 d	RBT	Some initial eye irritation. Quite, exhausted-looking, slight decrease in respiratory frequency.
142	30 min/d; 4 mo	RBT	No change in general condition. Some slight changes in blood parameters.

TABLE III-24. (continued)

Level (mg/m³)	Time	Species	Effects
142	7 d	RAT GPG	No histopathologic lesions or clinicopathologic changes.
71-142	4 d	GOAT	Increased rectal temperature, decreased food and water intake, and increased plasma cortisol levels. All returned to normal by the end.
41.6-142	8-24 h	MUS	Hypothermia, decreased food and water intake, weight loss, ~ 50% mortality. Pathological changes in the major organs. Survivors recovered their weight in ~ 2 wk.
50-100	5 d	RBT	No eye lesions.
49.7-92.3	16 h	RAT	Lethargy and heavy breathing by the end. Extensive organ damage in the one that died.
	100 h	RAT	Eye and nose irritation, rough hair. Recovered.
	100 h	GPG	Nose and eye irritation, rough hair. Recovered.
50	6 h/d; 6 mo	RAT	Decreased O_2 consumption. Increased erythrocyte and active neutrophil content, permeability of vessels, and respiration in heart and lung tissue.
14.2-42.6	?/d; 20 d	RBT	No change in respiration rate, WBC, RBC, hemoglobin, and autopsy findings. Slight weight decrease.
	4 h/d; 150 d	RBT	No change in general condition. Slight initial increase in free cholesterol.
28.4	48 h	MUS	Decreased food and water intake and rectal temperature. Weight loss.
28.4	90 d	MUS	Some mortality, survivors showing brain, liver, and lung abscesses. Survivors lost weight, had less endurance, and had instances of bronchopneumonia and hepatitis.

TABLE III-24. (continued)

Level (mg/m^3)	Time	Species	Effects
28.4	90 d	RAT	24% mortality, weight loss, lung pathology, and significant changes in many blood parameters.
28.4	1 h/d; 11 d	GPG	Fatigue, somnolence, dizziness, itching, and eye irritation. Decreased lipids in cerebral hemisphere and brain stem.
28.4	90 d	MKY	Weight loss. Changes in many blood parameters.
28.4	21 d	COW	No effect on feed intake, milk production, or heart rate. Slight lacrimation.
22.7	16 h	MUS	Slight restlessness. Normal autopsy.
	16 h	RAT	Slight initial restlessness.
14.2	5 d	MUS	Initial decrease in temperature. Decreased food and water intake, with some recovery by the end.
14.2	41 d	RAT	Also under cold stress, and showed decreased weight gain and food utilization. No changes in blood cells, plasma protein, or weight of liver and lungs.
14.2	4 d	GOAT	Initial decrease in food and water intake.
14	7 h/d; 14 d	DOG	Some coughing. Increased water consumption after each exposure. No change in blood picture or body weight.
12.1	17 d	PIG	NOEL for body weight gain and respiratory system.
10	3 h/d; 3 mo	RAT	Weight gain retarded. Changes in motor chronaxy. Irritation of mucous membranes of trachea and bronchi.
10	6 h/d; 4 mo	RAT	Retarded weight gain. Changes in some blood parameters (increased hemoglobin). O_2 consumption decreased by the end. Changes in lung and heart enzyme activity

TABLE III-24. (continued)

Level (mg/m³)	Time	Species	Effects
4.86- 9.36	6 h/d; 4 mo	RAT	Decreased liver glycogen and lipid levels, blood sugar, and erythrocytes. Increased blood lipids, hemoglobin, and leukocytes.
0.02	12 h/d; 3 mo	RAT	Changes in motor chronaxy. No change in weight. No pathological changes found in sacrificed animals.

SECTION IV

EXPERIMENTAL HUMAN INHALATION EXPOSURES

Table IV-1 describes acute laboratory human exposures to hydrogen sulfide. In the Summary, Table S-2 condenses all the information regarding experimental human exposure, from about 8,000 mg/m^3 to 0.008 mg/m^3. The American Conference of Governmental Industrial Hygienists gives 14.0 mg/m^3 as the time-weighted-average threshold limit value and 21 mg/m^3 as the short-term-exposure limit (ACGIH, 1980).

TABLE IV-1. HUMANS--ACUTE EXPERIMENTAL INHALATION EXPOSURE TO H_2S

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Mode of Exposure	No. of Test Subjects	No. of Controls	Duration and Frequency of Exposures	Total Length of Expt.	Effects	Reference and Rating
H_2S 8,165 (5,750)	Inhalation chamber	1 M, with 3 prior exposures to 1,420-4,700 mg/m^3 for several hours each	None	3 h 19 min	3 h 19 min	Pain in the nose, then eyes, then heavy continuous catarrh of both. Slight headache towards the end. Pale, trembling, and numbness. Afterwards, increased eye pain and catarrh, and roseola-type exanthemata (rose-colored rash) on 2 fingers.	Lehmann (1892) B-9
H_2S 7,554 (5,320)	Inhalation chamber	1 M, with 3 previous exposures to high levels	None	30 min	~ 2 h	Increasing irritation of the eyes and nose. Forced to leave the chamber for 2 min, after 20-min exposure, then returned. By the end: strong cough, heart palpitations, difficulty in breathing, violent pain in the eyes, dizziness, trembling, extreme fatigue, and in 10 min feeling of bilateral intracranial pressure. After exposure stopped, eye pains, heart palpitations, and pulse decreased. Fatigue, cranial pressure, diarrhea, bladder tenesmus, and lower body pains lasted until the next day.	Lehmann (1892) B-9
H_2S 7,540 (5,310)	Inhalation chamber	1 M, with 7 previous exposures to 1,420-8,165 mg/m^3 for several hours each	None	40 min	40 min	Irritation of the trachea, painful eyes, catarrh of nose and eyes, and a headache by the end which increased slightly after the exposure stopped.	Lehmann (1892) B-9
H_2S 5,297-7,001 (3,730-4,930)	Inhalation chamber	1 M, with 1 previous exposure to 3,124 mg/m^3	None	1 h 35 min	~ 2 h	Difficulty breathing, coughing, and irritation of the eyes, mouth, and nose. After exposure stopped, eyes were light-sensitive, painful, watering, and closed, with swollen eyelids and red conjunctiva, within 3 min. Pain in the eyes gone after 30 min. Fatigue, anorexia, light sensitivity, diarrhea, and bladder tenesmus continued until the next day.	Lehmann (1892) B-9

TABLE IV-1. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Mode of Exposure	No. of Test Subjects	No. of Controls	Duration and Frequency of Exposures	Total Length of Expt.	Effects	Reference and Rating
H_2S 3,499-5,832 (2,464-4,107)	Inhalation chamber	1 M, with 2 previous exposures to high levels	None	1 h 50 min	~ 4 h	Eye irritation, varying in intensity. Nasal catarrh. Difficulty breathing after 1 h 21 min. Considerable conjunctival congestion at the end. After exposure stopped, violent eye pains for 2 h, continuing through the night along with diarrhea, bladder tenesmus, fatigue, and bronchial catarrh.	Lehmann (1892) B-9
H_2S 4,700 (3,310)	Inhalation chamber	1 M, with a previous exposure to 1,420-2,130 mg/m^3 for 1 h	None	53 min	63 min	Increasing irritation of the nose, throat, trachea, and eyes. Headache 10 min after exposure stopped.	Lehmann (1892) B-9
H_2S 4,629 (3,260)	Inhalation chamber	1 M, with 4 previous exposures to 1,420-8,165 mg/m^3 for several hours each	None	2 h 25 min	2 h 25 min	After 45 min, tickling in throat and trachea. After 1 h 45 min, intense headache in the forehead and painful pressure in the eyes. No symptoms after exposure stopped.	Lehmann (1892) B-9
H_2S 2,982-3,976 (2,100-2,800)	Inhalation chamber	3 M, 1 with 9 previous exposures to high levels	None	0.5 h	0.5 h	Painful irritation of the nose and throat in 5-7 min. Very disagreeable by the end. Eye irritation. The 3 were about equally sensitive.	Lehmann (1892) B-9
H_2S 3,706 (2,610)	Inhalation chamber	1 M, with 2 previous exposures to 1,420 and 4,700 mg/m^3 for several hours each	None	46 min	46 min	Irritation of trachea and nose. Painful and watering eyes. No symptoms after exposure stopped.	Lehmann (1892) B-9

TABLE IV-1. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Mode of Exposure	No. of Test Subjects	No. of Controls	Duration and Frequency of Exposures	Total Length of Expt.	Effects	Reference and Rating
H_2S 3,550 (2,500)	Inhalation chamber	1 M, with 8 previous exposures to 1,420-8,165 mg/m^3 for several hours each	None	3 h 4 min	~ 5.5 h	Headache. Irritation of mucous membranes, leading to conjunctivitis. Symptoms disappeared in 2.5 h after exposure stopped.	Lehmann (1892) B-9
H_2S 2,982-3,266 (2,100-2,300)	Inhalation chamber	1 M, 27 y, strong and very healthy	None	52 min	~ 3 h	Immediate but temporary difficulty in breathing. Increasing irritation of the eyes (tearing, swelling, and secretions) and nose (secretions). Only slight nose catarrh 20 min after exposure stopped. Bladder tenesmus 2 h later, and diarrhea that night.	Lehmann (1892) B-9
H_2S 2,982 (2,100)	Inhalation chamber	1 M, with 9 previous exposures to 1,420-8,165 mg/m^3 for several hours each, 1 immediately before this expt.	None	2 h 38 min	~ 4 d	"Damp" headache, pains in the eyes and nose. By the end, strong headache and heavy tearing. These symptoms plus fatigue and sleeplessness continued for 2 d, decreased on the 3rd day. Subject recovered on the 4th day except for paleness, moodiness, and pain in the supraclavicular region.	Lehmann (1892) B-9
H_2S 2,982 (2,100)	Inhalation chamber	1 M, with 6 previous exposures to 1,420-8,165 mg/m^3 for several hours each	None	1 h	Several hours	After 5 min: irritation of throat. After 26-35 min: irritation of nose, larynx, and conjuctiva. After 1 h: headache on left side. Symptoms lasted several hours after the end of exposure.	Lehmann (1892) B-9

TABLE IV-1. (continued)

Compound(s) and Concentration(s) in mg/m³ (ppm)	Mode of Exposure	No. of Test Subjects	No. of Controls	Duration and Frequency of Exposures	Total Length of Expt.	Effects	Reference and Rating
H_2S 1,761-2,783 (1,240-1,960)	Inhalation chamber	1 M, with 4 previous exposures to high levels	None	3 h	~ 6.5 h	Increasing irritation of the eyes, mouth, and trachea, with coughing, for ~ 1 h. Eye irritation and sensitivity to light disappeared from 1.5 to 2 h, then returned along with difficulty in breathing. By the end, no complaints other than a small cough. Afterwards, a slight headache for 30 min, with fatigue, rhinitis, and bronchitis lasting for several hours.	Lehmann (1892) B-9
H_2S 1,988-2,130 (1,400-1,500)	Inhalation chamber	3 M, 1 with 9 previous exposures to high levels	None	1 h?	1 h?	Slight irritation of throat and eyes.	Lehmann (1892) B-9
H_2S 1,420-2,130 (1,000-1,500)	Inhalation chamber	1 M, young, strong, overweight	None	1 h	1+ h	Increasing irritation of the mucous membranes of the nose, throat, larynx, and conjuctiva. At the end, coughing. Symptoms continued "for a while" after exposure stopped.	Lehmann (1892) B-9
H_2S 2,059 (1,450)	Inhalation chamber	1 M, with 5 previous exposures to 1,420-8,165 mg/m³ for several hours each	None	3 h 56 min	~ 6 h	Headache after a few minutes. Irritation of the bifurcation of the trachea at the end of the exposure. Afterwards: conjunctivitis, strong nasal catarrh, and headache. Symptoms cleared in ~ 2 h, with tearing and headaches recurring later in the day	Lehmann (1892) B-9
H_2S 1,420-1,988 (1,000-1,400)	Inhalation chamber	1 M, with 5 previous exposures to high levels, 1 just before this expt.	None	3 h	~ 4 d	Increasing irritation of eyes and trachea, with burning pains, heavy salivation and mucous secretion, difficulty in breathing, and coughing for 1 h 45 min. By 1 h 55 min only nasal secretion remained, and was "absolutely well" by 2 h 15 min. Some recurrence of irritation at 2 h 37 min. Afterwards, painful, light-sensitive eyes and headache for 7 h, and some pain the next day. Bronchitis, rhinitis, and heavy conjunctivitis were diagnosed. Eye irritation remained after 4 d.	Lehmann (1892) B-9

TABLE IV-1. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Mode of Exposure	No. of Test Subjects	No. of Controls	Duration and Frequency of Exposures	Total Length of Expt.	Effects	Reference and Rating
H_2S 1,420-1,846 (1,000-1,300)	Inhalation chamber	1 M, with 6 previous exposures to high levels	None	1 h 23 min	1 h 23 min	Weak irritation of nasal mucous membranes.	Lehmann (1892) B-9
H_2S 1,704-1,804 (1,200-1,300)	Inhalation chamber	3 M, 1 with 9 previous exposures to high levels	None	1 h?	1 h?	Clearly disagreeable irritation of the eyes and throat.	Lehmann (1892) B-9
H_2S 994-1,278 (700-900)	Inhalation chamber	3 M, 1 with 9 previous exposures to high levels	None	1 h?	1 h?	Slight irritation of the throat and eyes.	Lehmann (1892) B-9
H_2S 284-568 (200-400)	Inhalation chamber	3 M, 1 with 9 previous exposures to high levels	None	1 h	1 h	No signs of irritation.	Lehmann (1892) B-9
H_2S 0.012-0.96	Inhalation	11, 16-55 y	Served as own controls	Exposures at each level repeated several times for unknown length of time	-	Odor not perceptible at 0.012-0.026 mg H_2S/m^3. Varying odor perception at 0.031-0.090 mg/m^3. Most perceived the odor at 0.10, and all did at 0.20-0.96 mg/m^3. Taking into account individual sensitivity, authors give the odor threshold of H_2S as 0.040 mg/m^3.	Loginova (1957) A-10
H_2S 0.08-0.5	Inhalation	14, 18-30 y, "practically healthy"	-	Not given, 1 test/d, daily for 3-4 d	-	Range of odor thresholds.	Baikov (1963) A-9
H_2S 0.27 (0.19)	Olfactometer with sniffing tubes	10	-	Not given	-	Odor threshold.	Williams et al. (1977) A-13

TABLE IV-1. (continued)

Compound(s) and Concentration(s) in mg/m^3 (ppm)	Mode of Exposure	No. of Test Subjects	No. of Controls	Duration and Frequency of Exposures	Total Length of Expt.	Effects	Reference and Rating
H_2S 0.15	Inhalation	8, 20-30 y	-	Not given	-	Threshold of odor objectionability (not odor detection).	Tonzetich and Ng (1976) B-9
H_2S 0.012-0.030	Inhalation	12	-	20-35 "observations"	-	Range of minimum concentrations at which the odor of H_2S was smelled.	Duan (1959) B-10
H_2S 0.013	Inhalation	3	Served as own controls prior to exposure	5 min, once, during min 15-20 of the expt.	60 min	Light sensitivity-related eye responses increased slightly more than at the 0.010 mg/m^3 exposure level, for the 40 min following exposure.	Duan (1959) B-10
H_2S 0.012	Inhalation	3, 18-28 y	Served as own controls prior to exposure	5 min, during min 15-20 of a 60-min expt., repeated on 3 days	180 min	Statistically significant increase in light sensitivity of the eye in the 40 min following exposure.	Baikov (1963) A-9
H_2S 0.010	Inhalation	3	Served as own controls prior to exposure	5 min, once, during min 15-20 of the expt.	60 min	Statistically significant increase in the light sensitivity-related eye responses by the 20th min, and for the following 40 min.	Duan (1959) B-10
H_2S 0.010	Inhalation	3, 18-28 y	Served as own controls prior to exposure	5 min, during min 15-20 of a 60-min expt., repeated on 3 days	180 min	No effect on light sensitivity of the eye.	Baikov (1963) A-9
H_2S 0.005-0.009 (3.39-6.44 ppb)	Inhalation chamber with face exposure only	998 M & F, ~ 20 y	-	15 s, once		Range of means of calculated odor thresholds from four test groups.	Adams et al. (1968) B-11
H_2S 0.008	Inhalation	3	Served as own controls prior to exposure	5 min, once, during min 15-20 of the expt.	60 min	Ability of eye to adapt to darkness not affected.	Duan (1959) B-10
H_2S 0.00067 (0.00047)	Odor test room	4 trained odor analysts	Served as own controls			Lowest concentration at which all the subjects positively recognized the odor.	Leonardos et al. (1969) A-11

SECTION V

EPIDEMIOLOGY

The Summary contains a discussion of the epidemiological studies and condenses the data into one table, Table S-3.

OCCUPATIONAL EXPOSURES

Data for occupational exposures to hydrogen sulfide are given in Table V-1. These studies are not particularly useful in establishing a range of concern for H_2S in automobile emissions because of the high levels in some, the probable presence of confounding factors in many (especially the petroleum and shale oil industry reports), and the possible inadequacies of the H_2S sampling and analysis procedures in others.

EXPOSURES OF THE GENERAL PUBLIC

Data from the two epidemiological studies of exposure of the general public are given in Table V-2. Weaknesses in study design considerably lessen the value of these reports in helping to determine a range of concern for H_2S.

TABLE V-1. STUDIES OF OCCUPATIONAL EXPOSURE TO H_2S

Compound(s) Concentration(s) mg/m^3 (ppm) Duration	Population Group			Effects	Remarks	Reference and Rating
	Description	Exposed	Controls			
H_2S < 28.4-> 852 (< 20-> 600) Air levels varied with location Also small amounts of CS_2, HCN, SO_2, and hydrocarbons	Shale oil plant workers	54 "acute cases"	None	Symptoms included: a sudden feeling of fatigue, especially in the legs; dizziness; and intense anxiety followed by unconsciousness with or without respiratory failure. Pains in back of head, dizziness, and sometimes nausea followed regaining consciousness. Symptoms subsided in half an hour except in some cases nystagmus (involuntary rapid eye movement) and Romberg's sign. Reflexes, coordination, eye movements, visual acuity, lungs, heart, blood, and blood pressure were normal.	Descriptive case study of clinical findings following relatively brief high concentrations of H_2S mixed with a variety of "gasoline vapors." As a group, exposures were poorly defined, but 7 case reports give exposure in great detail. Rates of symptoms and morbidity are not given for this group, although they would have been available. Therefore, the "risk" of unconsciousness, etc., is difficult to quantify from these data. There is some confusion between "acute," "subacute," and "chronic" exposures vs. effects. Subacute exposure, p. 260, is defined as extremely intense, but brief. This is in fact a mild acute exposure with subacute effects or symptoms. Acute effects may follow chronic exposure and vice versa.	Ahlborg (1951) B-9
H_2S 326.6 (230) Exposure lasted ~ 20 min	Worker in shale oil plant	1 M, 30 y	None	Found unconscious, froth about mouth, cramps in right arm, slow, shallow breathing, low blood pressure. He fully recovered in 6 d with no recurrence of symptoms over 2 y.		Ahlborg (1951) B-9

TABLE V-1. (continued)

Compound(s) Concentration(s) mg/m^3 (ppm) Duration	Population Group			Effects	Remarks	Reference and Rating
	Description	Exposed	Controls			
$H_2S \sim$ 40-185 (from 5 single grab samples)	Workers building a tunnel	50	-	In 9 mo, 163 instances of eye irritation were seen half an hour to several hours after descending into the tunnel: burning, grainy sensation, and haloes seen around lights. The symptoms were not pronounced while in the tunnel, but increased when workers came out: photophobia, lacrimation, broken blood vessels and tiny blisters on the cornea, and occasionally a light cough, irritation of the nose and pharynx, and nausea. The most exposed developed conjunctivitis and blepharitis. Large variability between individual responses.	Is exposure to H_2S reliable? No other gases were tested for. Workers in a similar tunnel, but with no H_2S detected, had no eye irritations. Reasonable descriptive information (intermittent exposure of 50 people), relevant to H_2S, but the issue of repeated and intermittent exposure is not discussed. Are the same persons equally at risk, are some repeatedly more sensitive than others, etc.?	Larsen (1944) A-9
$H_2S \sim$ 142 (~ 100) Apparently a one-time measurement for a problem which lasted several years	Coal miners	120	-	Symptoms of eye irritation occurred after several hours of exposure. Wide variability in severity of response. The light cases exhibited burning, conjunctival congestion, and lacrimation; symptoms disappearing in several hours. Severe cases exhibited intense burning, gritty sensation, conjunctival congestion, photophobia, blepharospasm, abundant lacrimation, and violent headaches lasting up to a week. No habituation was found, and possibly evidence of sensitization.	The one H_2S measurement done used an imprecise method, so the value is doubtful. From the range of clinical symptoms, it is evident that considerable variation in exposure levels occurred, which are essentially not measured. Without dose/duration measurement, it is difficult to see the value of this descriptive report to this project.	Deveze (1956) B-8

TABLE V-1. (continued)

Compound(s) Concentration(s) mg/m^3 (ppm) Duration	Population Group			Effects	Remarks	Reference and Rating
	Description	Exposed	Controls			
H_2S 13.7-36.6	Workers in a sugar beet processing plant	Not given	-	Eye complaints were seen, generally at the higher H_2S levels: pains, light sensitivity, tears, and headaches. Problems lasted several hours to days, and some workers had repeated eye pain.	No controls or comparison group. Apparently no other gases or compounds tested for.	Kranenburg and Kessener (1925) B-9
H_2S 15-35 (levels measured 4.3 h after exposure)	Accidental exposure of workers in a viscose plant while repairing a tank heater	9 M	-	4/9 complained of nausea, weakness, and pain in the chest, and were hospitalized. After 1 wk, no symptoms were found on internal, eye, neurological, and psychiatric examination and EKG. 5/9 were only briefly affected. All were examined 2 y later, and no after-effects were found.		Prouza (1970) B-5
H_2S 28.4 (20) Daily exposure level often exceeding this	Shale oil plant workers. Controls from the shale quarry	459 M	384 M	Frequency of nonoccupational disease the same in both groups. Plant workers reported 20% more fatigue, 5% more loss of appetite, and increased irritability. Slightly more headaches, loss of memory, and itching. 50% more complaints from conjunctivitis and 25% more from respiratory tract. Frequency of reported fatigue increased with length of employment and degree of exposure.	Attempt at non-concurrent prospective evaluation that falls down because of limited information on duration of exposure. Rates of symptoms are determined regardless of exposure duration and then stratified by those employed > or < 2 y. Since dramatic differences are present between these 2 groups, it would have been imperative to calculate person-month or person-year exposure to determine accurately differences in symptom frequency. The marked difference in fatigue, greater in Group I (exposed to H_2S) with light work load, suggests possibility of (1) interviewer bias or (2) drift of less physically fit worker to Group I. These potential biases are not discussed.	Ahlborg (1951) B-9

TABLE V-1. (continued)

Compound(s) Concentration(s) mg/m^3 (ppm) Duration	Population Group			Effects	Remarks	Reference and Rating
	Description	Exposed	Controls			
H_2S 7.1-14.2 (5-10) SO_2 not given "lower aliphatic compounds" Probably for at least several years	Gasoline desulfurization plant workers	20	20 workers of the same age from another dept. of the plant	There were 93 possible symptoms of irritation in exposed workers versus 23 for controls: 34 vs. 7, respiratory; 20 vs. 8, gastroenteritis; 30 vs. 3, eye; and 9 vs. 3, skin.	A survey of 40 workers in a petrol desulfurization plant, 20 of whom were at risk of toxicity to both H_2S and SO_2. Comparability of the 2 groups is not determined. Data suggests that low-level exposure to 5-10 ppm may have respiratory and mucous membrane effects. Duration of exposure needs to be determined.	Benini and Colamussi (1969) B-8
H_2S 7.1-14.2 (5-10) SO_2 not given 5-15 y (at least one worker had only intermittent exposure)	Gasoline desulfurization plant workers	30	-	Nine workers showed dermal affectations, 4 with papulopruritis and erythematous-wheal-like lesions (possibly indicating an allergic response), and 1 presented a syndrome of cutanea inveterata, pannicolopatia of the Besnier-Boek-Schaumann type and a miliary sarcoidosis of the lung of recent insurgence. Two of above 5 were sensitive to solution of H_2S in water as a patch test.	A descriptive survey of 30 workers, with no control or comparison groups.	Benini and Colamussi (1969) B-8
H_2S < 14.2 (< 10) Actual measurements weren't made; an alarm set for this level never went off	Workers in a heavy water plant	Not given	-	In 7 y, 123 cases of overexposure were seen: weakness, nausea, dizziness, headache, and nervousness. Includes several reports of apparently higher exposures, resulting in more severe symptoms and unconsciousness. A perforated eardrum and alcohol con-consumption both seem to increase the toxicity of H_2S.	This is a descriptive uncontrolled study in which there is no correlation of duration of exposure (e.g. distance from source, length of employment) and subsequent symptoms. Largely anecdotal account of the relationship of alcohol and H_2S, and the problem with perforated eardrums.	Poda (1966) B-4

TABLE V-1. (continued)

Compound(s) Concentration(s) mg/m^3 (ppm) Duration	Population Group			Effects	Remarks	Reference and Rating
	Description	Exposed	Controls			
H_2S 0-9.94 (0-7) 3 d, with at least the preceding 3 mo without H_2S exposure, before the plant started dedulfurization	Workers in a gasoline desulfurizing plant	13, 30-55 y	-	Slight and irregular changes were seen in the serum Fe and transferrin levels, and the elimination of the various fractions of urinary sulfates, in only some of the workers.	Its main use is the attempt at physiologic measurements to correlate with health outcomes. Only 13 persons were evaluated, therefore variable results and dubious conclusions. No dose-response relationship seen.	Benini and Colamussi (1970) B-6
H_2S 0.028-0.055	Breast-fed babies (1 mo-3 y) in a factory nursery whose mothers worked in the viscose spinning shop	Unknown no. out of 90 babies studied	Unknown no. out of 90 babies studied	Analysis was made of the air at the baby's nose while nursing. H_2S (and no CS_2) was found, emanating from the mothers' clothing. The babies were poorly developed, underweight, listless, anemic, pale, dyspeptic, and susceptible to more frequent and severe infectious diseases. They frequently vomited after feeding, and began walking and teething late. Babies with mothers in other shops were normal. When mothers were moved out of the viscose shops, the babies' development improved.	The hard data are limited, but this is a solid study. The observed correlation of spinning shop (but not other workshops) employment of the mothers with symptoms in the babies rules out most other likely causes.	Glebova (1960) B-8

TABLE V-2. EPIDEMIOLOGICAL STUDIES RELEVANT TO H_2S EXPOSURE

Compound(s) Concentration(s) mg/m^3 (ppm)	Population Group			Effects	Remarks	Reference and Rating
	Description	Exposed	Controls			
H_2S levels ranged from ~0.03-~0.43 (0.020->0.300), with levels of 0.020-0.125 ppm for 7 h one day, and >0.300 ppm for 1 h another day	Residents of affected areas of Terre Haute, Ind., May 18-June 15, 1964.	~ 41 (41 health/odor complaints received, 20 people interviewed by phone)	-	Numerous citizen complaints of nausea and vomiting, respiratory tract irritation, headaches, gastrointestinal complaints, shortness of breath, burning eyes, and disturbed sleep. Few were serious enough to warrant seeking medical treatment.	Weak cross-sectional study. Exposure data poorly defined with an almost a priori assumption of H_2S as the agent (what about SO_2, particulates, etc.). Only the 20 individuals who originally complained were interviewed, without any controls or appropriate comparison group. Standard epidemiologic measures were not used. Absentee rates were derived from hospitals; what of susceptible individuals (i.e., children, school rates, etc.)? Information is suggestive but sampling is inadequate, controls are absent, and this must cast some doubt on the conclusions.	U.S.P.H.S. (1964) A-6
H_2S 0.005-0.300 Hydrocarbons	Residents of 113 apartments using unpurified natural gas for heating	Not given	Not given, residents in same city, but without gas heat	Those in households with $\geq$ 0.05 mg/m^3 complained of headaches, weakness, nausea, and vision problems. Reactions of sampling crew indicate that the odor threshold for H_2S in natural petrolic gas was 0.01-0.03, and for free H_2S was 0.04. For a period of 2 y, the morbidity per 1,000 residents was ~ 50% higher in the exposed group. Morbidity rates were higher in the test group for the < 3, 20-49, and 50-59 y age groups.	The study is fascinating, but raises serious questions of the comparability between the two regions. Although the sex distribution is similar and age-specific rates are considered (note that the summary difference - 52% - is not age-adjusted), no comparability of socioeconomic status is present. In view of the 40% increase in infectious disease in the exposed group, this difference is a significant one, and perhaps the major explanation for the difference in morbidity rates. It is not uncommon to see a 40% increase among the poor or disadvantaged (i.e., those without gas heat?). Also note the varying widths of the age ranges used, particularly the wide 20-49 y group, where most of the morbidity was found.	Loginova (1957) B-10

BIBLIOGRAPHY*

4-127 ACGIH, American Conference of Governmental Industrial Hygienists Committee on TLVs. 1971. Documentation of Threshold Limit Values for Substances in Workroom Air, ed. 3. ACGIH. Cincinnati, Ohio. pp. 11-12.

B--. The usefulness to this review is limited only by the goal of intermittent (40 h/wk) exposure. Several sources reported eye effects at ≤ 20 ppm, so the TLV was set below that, at 10 ppm.

4-340 ACGIH, American Conference of Governmental Industrial Hygienists TLV Airborne Contaminants Committee. 1978. TLV's Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1979. ACGIH. Cincinnati, Ohio.

D--. The time-weighted-average TLV was 15 mg/m^3 and the short-term-exposure limit was 27 mg/m^3.

5-175 ACGIH, American Conference of Governmental Industrial Hygienists TLV Airborne Contaminants Committee. 1980. TLVs Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1980. ACGIH, Cincinnati, Ohio. p. 20.

A--. The time-weighted-average TLV value for H_2S is 14 mg/m^3 and the tentative short-term-exposure limit is 21 mg/m^3.

4-310 Adams, D. F., F. A. Young, and R. A. Luhr. 1968. Evaluation of an Odor Perception Threshold Test Facility. TAPPI 51(3):62A-67A.

B-11. Four groups of subjects, totaling 998, had calculated thresholds of detection for H_2S from 3.39 ppb to 6.44 ppb. Differences between groups were seen. Extensive data manipulation was done.

4-121 Adelson, L., and I. Sunshine. 1966. Fatal Hydrogen Sulfide Intoxication. Arch. Pathol. 81:375-380.

D-5. Case report of three fatalities due to inhalation of sewer gas containing unknown but high levels of H_2S. It was rapidly fatal due to paralysis of the central nervous system.

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* MRI document acquisition numbers are given in the left margins.

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4-122 Ahlborg, C. 1951. Hydrogen Sulfide Poisoning in Shale Oil Industry. AMA Arch. Ind. Hyg. Occup. Med. 3:247-266.

B-9. Case reports and large group studies of workers exposed to 20-600 ppm H_2S. Symptoms range from conjunctivitis to neurasthenic symptoms and unconsciousness. Studies are poorly designed and confounded by the presence of other gases.

4-341 AIHA, American Industrial Hygiene Association. 1962. Hygienic Guide Series; Hydrogen Sulfide. American Industrial Hygiene Association, Akron, Ohio. 2 pp.

D--. Brief review.

4-001 Aitbaev, T. K., V. M. Almaniyazova, N. A. Strelyukhina, A. S. Abylkasymova, and U. A. Rysbekova. 1976. Combined Effect of Some Industrial Gases. Zdravookhr. Kaz. No. 1:69-72 (Russ).

B-9. Albino rats were exposed to H_2S at the MAC level (the MAC in 1972 was 10 mg/m^3) for 6 h/d for 4 mo. A slight decrease in body weight gain, fluctuations in peripheral blood values, and an ultimate decrease in 2 indexes of O_2 consumption were seen. An interaction study of H_2S, HF, and SO_2 was done, indicating summation-type action.

4-195 Aizenshtad, V. S., E. G. Dolmatova-Guseva, V. P. Perkhurova, A. V. Shtifel'man, L M. Bogomolova, and S. M. Nerubai. 1971. Work Hygiene and the State of Workers' Health in the Production of Carbophos. Gig. Tr. Prof. Zabol. 15(3):49-51 (Russ).

D--. Workers engaged in the production of carbophos (malathion) were exposed to dimethyldithiophosphoric acid, diethyl maleate, the product, methanol, and xylene as well as to H_2S. The latter four compounds exceeded their maximum allowable concentrations (MAC) in 11 to 17% of the air samples. Carbophos exceeded its MAC, however, in 35% of the cases. Acute conjunctivitis and dermatitis were possibly due to H_2S since the incidence of these maladies decreased from 18 to 2 persons when the H_2S concentration was reduced by control measures.

4-129 American Industrial Hygiene Association. 1962. AIHA Hygienic Guide Series. Hydrogen Sulfide. American Industrial Hygiene Association, Akron, Ohio. 2 pp.

D--. Very authoritative summary, recommending a maximum atmospheric concentration (8 h) of 20 ppm.

4-130 American Petroleum Institute Toxicological Review. 1948. Hydrogen Sulfide. Department of Safety, A.P.I., New York, New York. 6 pp.

D--. A review of H_2S poisoning: occurrence, effects, treatment, and precautionary measures. The threshold is below 50 ppm.

4-131 American Standards Association. 1941. Allowable Concentration of Hydrogen Sulfide. ASA Z37.2-1941, A.S.A., New York, New York. 6 pp.

D--. Old, very short review giving 20 ppm as the MAC for 8 h.

4-196 Andreescu, M. 1970. Comparative Investigation of Some Adenocarcinomas of the Mammary Glands in Mice of Lines WHT/Ht, A2G and R III, Developed Under the Influence of Harmful Exogenous Factors. Nats. Kongr. Onkol., Sb. Dokl. 1st; Vol. 3. pp. 43-48 (Fre).

C--. Three strains of mammary-tumor-bearing mice were exposed to one of several factors, including an unknown level of H_2S. The character of the tumor chromosomes didn't change.

4-273 Andrew, F. D., R. A. Renne, and W. C. Cannon. 1979. Toxic Effects of Geothermal Effluents: Reproductive Toxicity Testing for Effects of H_2S in Rats. In: Pac. Northwest Lab. Annu. Report 1979 DOE. Assist. Secr. Environ. Pt. 1: Biomedical Science. pp. 276-278.

D-9. An abstract of work in progress. Short, repeated exposure to 220 ppm H_2S did not induce dominant lethal effects during spermatogenesis in male rats or embryotoxicity in the progeny of female rats. Minor rib anomalies were found in some of the fetuses (authors' summary).

4-197 Andriasian, G. K. 1969. Certain Side Effects of Hydrogen Sulfide Baths of Matsesta. Vestn. Dermatol. Venerol. 43(1):72-73 (Russ).

D--. The incidence and case histories of skin problems in people using baths containing 100-150 mg H_2S/L are described.

4-240 Anichkov, S. V., and M. L. Belen'kii (R. Crawford, Trans.). 1963. Pharmacology of the Carotid Body Chemoreceptor. The Macmillan Co. New York, New York. pp. 49-60.

D--. A review of the mechanisms of toxicity of sulfides, including H_2S, through several routes of exposure. Inhalation of H_2S caused erythrocytosis and hyperglycemia, involving reflexes from the carotid chemoreceptors.

4-132 Anonymous. 1952. Four Workers Overcome by Hydrogen Sulfide when Digging in Marshy Land. Occup. Health 12:39.

C-6. Case report of exposure to 300-500 ppm H_2S causing unconsciousness, nausea, and stomach pains.

4-133 Anonymous. 1966. Gas Hazards in Underground Tanks and Wells. Mich. Occup. Health 11:1-2.

C-7. Case reports of two accidental poisonings, in one of which $\geq$ 1,000 ppm H_2S was fatal.

4-003 Anonymous. 1978. Hydrogen-Sulphide Poisoning. Lancet 1(0054): 28-29.

D--. A short review of the occurrence and toxicity of H_2S. Includes fatal and nonfatal case reports.

4-343 Anonymous. 1981. Deaths Associated with Liquid-Manure Systems--United States. Morb. Mortal. Wkly. Rep. 30(13):151-152, 157.

D--. A report of 8 deaths and 2 cases of near-fatal illness, in 3 separate accidents. H_2S was believed to be the most likely causative agent along with decreased O_2 and increased CO_2. Investigation after one accident led to an estimate of $\geq$ 570 ppm H_2S at the time of the incident. No symptoms are described.

4-005 Arimatsu, T., and K. Kesado. 1978. Intoxication due to Hydrogen Sulfide During the Application of a Mixture of Lime-Sulfur and Primary Calcium Phosphate. Nippon Noson Igakkai Zasshi (Jap. J. Rural Med.). 27(3):452-453 (Japan).

C-7. A woman accidentally exposed to $\sim$ 5,000 ppm H_2S lost consciousness for 4 h, and suffered nausea and vomiting lasting 4 d and leukocytosis.

4-134 Aufdermaur, F., and O. Tönz. 1970. Poisoning of Children by Liquid Manure Gas When Using "Rustic" Latrines. Schweiz. Med. Wochenschr. 100:894-896 (Ger).

C-8. Case reports of 3 accidental poisonings with unconsciousness, coma, convulsions, irritation of mucous membranes, lung edema, and leukocytosis.

4-241 Aves, C. M. 1929. Hydrogen Sulphide Poisoning in Texas. Texas State J. Med. 24:761-766.

C--. An analysis of the air in some Texas oilfields found 2,000-140,000 ppm H_2S. Several acute, fatal cases are reported. Chronic effects included: slow healing of minor skin wounds, eye irritation, weight loss, and insomnia. Increased inflammation of air passages or respiratory infections was not seen. Specific H_2S concentrations for the case reports are not given.

4-285 Baikov, B. K. 1963. Maximum Permissible Concentration of Carbon Disulfide and Hydrogen Sulfide when Present Together in the Atmosphere. Gig. Sanit. No. 28:3-8 (Russ).

A-9. The odor threshold for H_2S was found to be 0.014-0.03 mg/m^3. Light sensitivity of the eye wasn't affected by 0.01 mg/m^3. Effects when combined with CS_2 were additive and possibly synergistic, respectively.

4-006 Baltaitis, V., Y. M. Markovich, I. F. Yarembash, and A. S. Grebtsova. 1975. Razrab. Mestorozhd. Polezn. Iskop. (Kiev). 39:158-166 (Russ).

D--. CO and HCl were released in high concentrations from burning poly(vinyl chloride). Additional combustion products measured were NO_x, SO_2, $COCl_2$, HCN, and H_2S.

4-007 Barilyak, I. R., and I. A. Vasil'eva. 1974. Antimitotic and Cytogenetic Activity of Small Concentrations of Carbon Disulfide and Hydrogen Sulfide. Tsitol. Genet. 8(2):126-129 (Russ).

D--. The exposure of rats to 10 mg H_2S/m^3 and CS_2 (whether that is each or the total concentration is unclear) for 70-90 d caused decreased mitotic activity of liver and kidney embryonic cells and increased incidence of aneuploidy and structural chromosomal aberrations in adult bone marrow cells.

4-008 Barilyak, I. R., I. A. Vasil'eva, and L. P. Kalinovskaya. 1975. Effect of Small Concentrations of Carbon Disulfide and Hydrogen Sulfide on the Intrauterine Development of Rats. Arkh. Anat. Gistol. Embriol. 68(5):77-81 (Russ).

D--. Exposing pregnant rats to H_2S and CS_2 (at 10 mg/m^3 [not clear whether this is the concentration of each component]) under conditions of the viscose industry for prolonged periods was embryolethal at the pre- and post-implantation stage. Abnormalities, primarily of the urogenital and bone systems, were seen in a small number of the embryos. Blood formation and ossification were disturbed in the embryos, and there were severe changes in parenchymatous cells of the liver and kidneys of the rat embryos.

4-278 Barthelemy, M. L. 1939. Ten Years' Experience with Industrial Hygiene in Connection with the Manufacture of Viscose Rayon. J. Ind. Hyg. Toxicol. 21:141-151.

C-6. Descriptive study correlating conjunctivitis and visual symptoms with avg. concentrations of H_2S (12-41 mg/m^3), CS_2 (63-162 mg/m^3), and H_2SO_4 (31-55 mg/m^3). Controls are absent. The authors assume that H_2S is the culprit because workers in another department exposed to 400 mg CS_2/m^3 for a few hours had no such symptoms. If this assumption is accepted, the threshold level for H_2S for conjunctivitis is < 25 mg/m^3. The authors believe that the presence of CS_2 and H_2SO_4 create a hypersensitivity of the conjunctiva and cornea to H_2S.

4-290 Basmadzhieva, K., Z. Rashev, and M. Argirova. 1969. Adaptation of Rats to Low Atmospheric Concentrations of Hydrogen Sulfide and Phenol. Khig. Zdroveopazvane. 12(1):33-37.

D--. Rats exposed to a mixture of H_2S (0.03 mg/m^3) and phenol (0.06 mg/m^3) for 3 mo exhibited a general adaptation syndrome.

4-010 Baynes, C. J., and A. G. Scott. 1977. Estimating the Probability of a Lethal Exposure During an Episodic Release of a Toxic Gas. In: Proc., Annu. Meet. - Air Pollut. Control Assoc., 70th (Vol. 4), 77-445, 14 pp.

D--. A theoretical, statistical model for predicting exposure is described. It may be relevant to estimating margins of safety to allow for fluctuations.

4-136 Beasley, R. W. R. 1963. The Eye and Hydrogen Sulfide. Br. J. Ind. Med. 20:32-34.

C-5. Description of the same cases as in Carson (1963).

4-011 Beck, J. F., F. Cormier, and J. C. Donini. 1979. The Combined Toxicity of Ethanol and Hydrogen Sulfide. Toxicol. Lett. (AMST). 3(5):311-314.

C-12. An interaction study. Rats injected with ethanol had shorter times to unconsciousness when exposed to 800 ppm H_2S than rats without alcohol exposure.

4-012 Becker, B. 1978. Hazards Through Hydrogen Sulfide--G 11. Zentralbl. Arbeitsmed. Arbeitsschutz. Prophyl. 28(8):224-226 (Ger).

D--. A review with no references for occupational physicians.

4-199 Benini, F., and V. Colamussi. 1969. Various Chronic or Relapsing Skin Manifestations, Observed in Refinery Workmen Exposed to Long Term Action of Hydrogen Sulfide. Arcisp. S. Anna Ferrara 22:973-983 (Ital).

B-8. Workers were exposed to unknown levels of SO_2 and "lower aliphatic compounds" as well as 5-10 ppm H_2S. Various respiratory symptoms, eye irritation, and skin sensitization were found.

4-198 Benini, F., and V. Colamussi. 1970. Possibility of Absorption of Hydrogen Sulfide in Workers Employed in a Confined Place. Arcisp. S. Anna Ferrara 23(6):541-548 (Ital).

B-6. Environmental conditions (including H_2S, 0-7 ppm), serum Fe and transferrin levels, and the elimination of various fractions of urinary sulfate were measured in 13 workers in a gasoline desulfurizing plant. Only slight changes were found. Results were too variable (irregular) to be useful--i.e., there was no dose-response relationship.

4-200 Benson, F., and T. Karlsson. 1972. Hydrogen Sulfide Intoxication--Toxicity and Therapy. Lakartidningen 69(6):627-631 (Swe).

C--. A good review with two case reports of acute poisoning and treatment.

4-013 Bersch, W., U. Meinhof, G. Ule, H. Berlet, and A. M. Thiess. 1974. Proceedings: Pathomorphologic and Pathochemical Findings in Acute H_2S-Poisoning in Man. Verh. Dtsch. Ges. Pathol. 58:502 (Ger).

D--. Review.

4-323 Berushvili, Ts. A. 1980. Hygienic Evaluation of Hydrogen Sulfide-Containing Hot Springs Used in Hot Water Supply Systems. Gig. Sanit. No. 6:11-13 (Russ).

D--. Tests with these waters in guinea pigs and white rats were concerned only with the effects on the skin and effects after ingestion.

4-014 Biesold, J., M. Bachofen, and H. Bachofen. 1977. Pulmonary Edema due to Hydrogen Sulfide. Lung 154(2):146.

D-7. A morphological examination of the lungs of the victim of a fatal H_2S inhalation.

4-201 Bittersohl, G. 1971a. On Relationships in Action Between Carbon Disulfide and Hydrogen Sulfide. Med. Lav. 62(12):554-556.

D--. A hypothesis for the mechanisms of action and interaction of H_2S and CS_2.

4-202 Bittersohl, G. 1971b. Toxical Effect of Hydrogen Sulfide. Z. Gesamte Hyg. 17(5):305-308 (Ger).

D--. Mechanism review.

4-624 Blaser, E. 1946a. A Contribution to the Study of Hydrogen Sulfide Poisoning of Animals by Manure Gases. Schweiz. Arch. Tierheilkd. 88:401-413 (Ger).

D-8. Case report of deaths of two horses due to sewer gas. No analysis of constituents. Pathological-anatomical study of the bodies is reported.

4-324 Blaser, E. 1946b. A Contribution to the Study of Hydrogen Sulfide Poisoning of Animals by Manure Gases. Schweiz. Arch. Tierheilkd. 88:433-446 (Ger).

C--. Small numbers of rabbits, guinea pigs, mice, and doves were exposed to high levels of H_2S (0.24 or 0.40%) and NH_3 (0.10% or 0.24-0.362%) together for several hours. Histological examinations were done showing effects in the lungs, heart, liver, kidney, and spleen.

4-016 Blaxland, J. D., J. Shemtob, G. H. Francis, and G. E. Jones. 1978. Mortality in a Battery Laying House Attributed to the Presence of Noxious Gases from Slurry. Vet. Rec. 103(11):241-242.

D-7. H_2S levels may have reached as high as 90 ppm near the most exposed chickens, causing increased mortality. On autopsy, several abnormalities were found, including lung edema. Confounded by the possibility of other factors present.

4-017 Booras, S. G. 1974. Hydrogen Sulfide Health Effects and Recommended Air Quality Standard. PB 233-843, National Technical Information Service, U.S. Dept. of Commerce, Springfield, Virginia. 34 pp.

A--. The thrust of this review is similar to that of the current project. Review contains atmospheric H_2S data, acute and subacute poisoning of humans, and a recommended air quality standard of 0.015 mg/m^3.

4-138 Breysse, P. A. 1961. Hydrogen Sulfide Fatality in a Poultry Feather Fertilizer Plant. Am. Ind. Hyg. Assoc. J. 22:220-222.

C-6. The victim was apparently exposed to 2,000-4,000 ppm H_2S for up to 15 min.

4-139 Breysse, P. A. 1970. Three Men Rendered Unconscious While Working in Sewer Manhole. Occup. Health Bull. 25:1-3.

D-4. The authors believe the effect (unconsciousness after 2 min) was due to H_2S, though there are very few data. Several days after the incident, no H_2S was detected.

4-140 Brown, K. E. 1969. Some Toxicological Problems in a Rubber Industry. Med. J. Aust. 1:534-538.

C-4. H_2S level of about 100 ppm caused unconsciousness, with apparent full recovery in 1 day.

4-019 Buevich, V. A., and G. M. Mel'nikova. 1973. Biophysical Aspects of Muscle Tone. Dokl. Akad. Nauk. SSSR, Ser. Biol. 212(2):490-491 (Russ).

D--. The muscle tone of workers exposed to both H_2S and CS_2 was compared to those of workers not exposed to sulfur compounds at the same plant and of persons with influenza.

4-141 Bulatova, F. D., L. I. Geller, I. S. Genadinnik, and V. A. Sukhanova. 1968. Influence of Products from the Conversion of High-Sulfur Petroleum on the Extent and Clinical Manifestations of Chronic Bile Duct Disease. Gig. Tr. Prof. Zabol. 12:22-26 (Russ).

D--. Oil refinery workers exposed to hydrocarbons and H_2S at concentrations 4-6 times the maximum allowable concentrations showed a higher incidence of diseases implicating the bile duct than did railroad depot workers or lathe operators. Inflammatory signs in patients with chronic cholecystitis were mild. Functional disorders of the gall bladder and gastrointestinal tract and deranged liver and gastric secretory functions were prevalent.

4-293 Burnett, W. W., M. Grace, W. F. Hall, and E. G. King. 1975. The Problem of Hydrogen Sulfide Poisoning. Alberta Med. Bull. 40:68-69.

D--. A brief discussion of H_2S exposure in the petrochemical industry in Alberta, Canada; symptoms and treatment, but no concentrations.

4-020 Burnett, W. W., E. G. King, M. Grace, and W. F. Hall. 1977. Hydrogen Sulfide Poisoning: Review of 5 Years' Experience. Can. Med. Assoc. J. 117(11):1277-1280.

C-8. Review of observed symptoms but no exposure data. The acute problems in the 221 cases studied were coma, dysequilibrium, and respiratory insufficiency with pulmonary edema. Survivors had no apparent long-term adverse effects.

4-142 Carson, M. B. 1963. Hydrogen Sulfide Exposure in the Gas Industry. Ind. Med. Surg. 32:63-64.

D-5. An account of five men with varying exposures (intermittent, over 4 wk) to an atmosphere containing H_2S and NH_3 in unknown concentrations. The three affected men complained of blurred vision, seeing colored rings around lights, and "gritty" eyes. Symptoms lasted only a day.

4-022 Chelikanov, K. N. 1978. Nature of the Combined Action of Carbon Disulfide, Hydrogen Sulfide, and Sulfur Dioxide When All are Present in the Air. Nauch. Tr. Ryazan. Med. In-t. No. 63:45-47 (Russ).

D--. Rats were exposed to a mixture of 0.005 mg CS_2/m^3, 0.003 mg H_2S/m^3, and 0.05 mg SO_2/m^3 or one of 0.00172 mg CS_2/m^3, 0.0026 mg H_2S/m^3, or 0.0169 mg SO_2/m^3. Changes were seen in the blood chemistries of these two groups compared to the controls. The gases were said to have a summation effect showing neither synergism nor antagonism.

4-023 Chepikova, N. R. 1974. Effect of Hydrogen Sulfide Baths on the Resistance of Animals to Acute Hypoxia. Vop. Kurortol., Fizioter. Lech. Fiz. Kul't. No. 1:70-76 (Russ).

D--. One-time bathing of mice in solutions containing 25 or 50 mg H_2S/L increased their resistance to experimentally induced hypoxia. The increase in resistance was explained by mobilization of blood from the spleen to increase the respiratory capacity of the blood.

4-143 Chertok, O. M. 1968. Acute Poisoning with Hydrogen Sulfide. Zh. Nevropatol. Psikhiatr. 68:381-384 (Russ).

C-5. The 29 workers studied were divided into three groups of light, moderate, and severe intoxication, and the symptoms are described. However, no actual exposure information is given. The group with light exposure experienced headaches, vertigo, a feeling of fainting, difficulty in breathing, nausea and vomiting, photophobia, pain in the eyes, and a short loss of consciousness sometimes with convulsions.

4-025 Combaz, M. 1976. Gas Intoxications in a Petroleum Refinery (1969 to 1974). Staub - Reinhalt. Luft. 36(1):15-19 (Fre).

D-8. Among 40 instances of gas intoxication within 5 y at a petroleum refinery employing an average 2,000 people, 15 instances were due to the presence of H_2S. Toxicity due specifically to H_2S and H_2S concentrations causing the intoxications were not stated.

4-266 Comberg, G. 1965. Stall Climate as a Performance Factor in Animal Confinement. Tierzuechter. 17(14):81-82 (Ger).

D--. Review of animal exposure to H_2S, NH_3, and CO_2 in closed barns.

4-026 Cordasco, E. M., and E. D. Stone. 1973. Pulmonary Edema of Environmental Origin. Chest 64(2):182-185.

D--. A review of treatment used in cases of pulmonary edema caused by chemical agents, including H_2S. Includes a very brief summary of the effects of H_2S, as low as 50 ppm being toxic.

4-144 Cralley, L. V. 1942. The Effect of Irritant Gases Upon the Rate of Ciliary Activity. J. Ind. Hyg. Toxicol. 24:193-198.

D-6. Ciliary activity in freshly excised rabbit tracheal tissue was completely stopped, without recovery in air, by exposure to 600 ppm H_2S for 5 min.

4-027 Curtis, S. E., C. R. Anderson, J. Simon, A. H. Jensen, D. L. Day, and K. W. Kelley. 1975. Effects of Aerial Ammonia, Hydrogen Sulfide and Swine-House Dust on Rate of Gain and Respiratory-Tract Structure in Swine. J. Anim. Sci. 41(3):735-739.

C-10. H_2S, alone at 8.5 ppm or at 2 ppm in combination with 50 ppm NH_3, had little effect on the growth rates of three pigs, exposed for 17-19 d.

4-028 Dalgaard, J. B., F. Dencker, B. Fallentin, P. Hansen, B. Kaempe, J. Steensberg, and P. Wilhardt. 1972. Fatal Poisoning and Other Health Hazards Connected with Industrial Fishing. Br. J. Ind. Med. 29(3):307-316.

D-7. Several case reports of fatal and nonfatal accidents, probably due to the presence of noxious gases (H_2S, NH_3, HCHO, CO_2) and/or low oxygen levels. The H_2S levels found in the holds of several ships (not done at the time of the accidents) ranged from 3 to > 2,000 ppm.

4-145 Davenport, S. J. 1945. Hydrogen Sulfide Poisoning as a Hazard in the Production of Oil. Bureau of Mines, U.S. Dept. of the Interior, Information Circular 7329. 10 pp.

C--. General discussion of H_2S, its toxicology, and workplace precautions. Review includes several cases of poisoning in mining operations.

4-029 Demaret, D., and J. Fialaire. 1974. Hydrogen Sulfide Poisoning in a Natural Gas Refinery. J. Eur. Toxicol. 7(1):32-36 (Fre).

C-9. Workers in natural gas refineries experienced a wide range of symptoms: light (euphoria, staggering, colored vision), moderate (unconsciousness, headache, nausea, respiratory irritation), and severe (unconsciousness and coma, polypnea, tachycardia, convulsions). All eventually recovered. No levels of H_2S in the air were given, only in the gas being refined (14.5% - 145,000 ppm).

4-258 Denmead, C. F. 1962. Air Pollution by Hydrogen Sulfide from a Shallow Polluted Tidal Inlet, Auckland, New Zealand; Paper No. 4, In: Proceedings of the First Technical Session of the Clean Air Conference, University New South Wales, Auckland, New Zealand. 17 pp.

D--. Microbial action in polluted waters caused atmospheric H_2S levels of < 1 ppm from ~ 2,000-6,000 ft away. Public complaints of paint blackening and odor were blamed on the H_2S present.

4-235 Devèze [no initial]. 1956. Hydrogen Sulfide, a Pathological Factor in Coal Mining. Evidence Drawn from 100 Observations. Rev. Med. Minière. 9(32-33):14-18 (Fre).

B-8. In over 4 years, 120 workers complained of eye irritation of widely varying severity. A doubtful spot value of 100 ppm H_2S was measured.

4-031 Dirnhofer, R. 1977. The Morphology of Hydrogen Sulfide Poisoning. Beitr. Gerichtl. Med. 39:145-159 (Ger).

C-9. Case report of a fatility due to cardiac failure several days after sewer gas intoxication. There were alterations of the central nervous system and serious disturbances in the cell metabolism of the myocardium and liver. Authors believe the effects were due to H_2S (unknown level). The report implies that any cardiac patient (or atherosclerotic) who already has myocardial cell problems belongs to a susceptible group.

4-326 Division of Industrial Hygiene, U.S. Public Health Service. 1941. Hydrogen Sulfide: Its Toxicity and Potential Dangers. Public Health Rep. 56:684-692.

C--. Review of the toxicology of H_2S and the pathology of poisoning. Effects of exposure are similar to other reports.

4-033 Donham, K. J., M. Rubino, T. D. Thedell, and J. Kammermeyer. 1977. Potential Health Hazards to Agricultural Workers in Swine Confinement Buildings. J. Occup. Med. 19(6):383-387.

D-7. The H_2S levels in 13 different swine confinement units varied with time, from 0 to 10 ppm, along with dust and high levels of NH_3, CO, and CO_2. The 11 workers and 35 veterinarians interviewed complained of several adverse symptoms, including coughing and eye irritation.

4-262 Dougherty, R. W., R. Wong, and B. E. Christensen. 1943. Studies of Hydrogen-Sulfide Poisoning. Am. J. Vet. Res. 4:254-256.

D-6. The toxicity of H_2S gas to dogs and sheep, introduced through the rectum, was affected by the amount of CO_2 in the air breathed. Artificial respiration wasn't sufficient to keep a guinea pig alive after receiving a toxic H_2S dose.

4-146 Duan Fyn-Zhui. 1959. Data for Determining the Maximum Permissible Concentration of Hydrogen Sulfide in the Atmospheric Air. Gig. Sanit. 10:12-17 (Russ); English translation in: USSR Literature on Air Pollution and Related Occupational Diseases. A Survey. B. S. Levine (trans.). Vol. 5, U.S. Public Health Service, Washington, D.C. pp. 66-73.

A-10. The minimum concentration at which H_2S odor was detected was 0.012-0.030 mg/m^3. Light sensitivity of the eye wasn't affected at 0.008 mg/m^3, and was affected at 0.01-0.013 mg/m^3. Motor chronaxy studies (indicating the functional state of the brain cortex) and autopsies show definite changes in rats exposed to 10 mg H_2S/m^3 for 12 h/d, 3 mo. Those exposed to 0.02 mg/m^3 showed weaker symptoms. Authors suggest 0.008 mg H_2S/m^3 as the allowable daily avg. concentration.

4-035 Dubrovskaya, F. I., M. S. Katsenelenbaum, Y. K. Yushko, S. A. Ipatova, and R. P. Vlasov. 1976. Hygienic Evaluation of Air Protection Measures at the Volga Industrial Complex. Gig. Aspekty Okhr. Okruzhayushchei Sredy 123-127 (Russ).

D--. Exposure of children living in the vicinity of the Volga industrial complex to HCHO, SO_2, aldehydes, H_2S, and NH_3 increased morbidity. Effects were not ascribed to particular substances and the levels of the compounds were not stated except that they exceeded their limits.

4-037 Dvoriantseva, M. V. 1973. Treatment of Chronic Dermatoses Using Hydrogen Sulfide Baths from Sokolovogorskii Spring. Vopr. Kurortol. Fizioter. Lech. Fiz. Kult. 38(3):252-254 (Russ).

D--. Mineral baths containing NaCl and 20-32 mg H_2S/L improved conditions of eczema and psoriasis in 88.1 and 84.9% of the patients. Improvement was seen in 81.5% of the patients with neurodermatitis.

4-036 Dwornicki, J. 1979. Effect of Experimental Poisoning with Hydrogen Sulfide on the Cardiac Muscle. Bromatol. Chem. Toksykol. 12(3):256-265 (Pol).

C--. Exposing rabbits for 1 h/d for 14 d to 100 mg H_2S/m^3 caused disorders of rhythm, mineral balance, and enzyme activity in their hearts.

4-039 Elebekova, R. S., T. K. Aitbaev, and V. M. Almaniyazova. 1976. Functional State of the Liver under the Chronic Combined Effect of Hydrogen Fluoride and Hydrogen Sulfide. Tr. Nauchn.-Issled. Inst. Kraev. Patol. (Alma Ata). 29(Part 1?):27-31 (Russ).

B-11. Good work. Omits explicit statement of "no observed toxic signs." Little experimental detail is given, but appears consistent internally. Rats were exposed to 5 or 10 mg H_2S/m^3 for 6 h/d, 6 d/wk, for 4 mo. Changes in liver function and peripheral blood values were seen. Interaction study with HF was also done. Includes an extensive review of other Russian work with H_2S (both oral and inhalation routes).

4-040 Elovaara, E., A. Tossavainen, and H. Savolainen. 1978. Effects of Subclinical Hydrogen Sulfide Intoxication on Mouse Brain Protein Metabolism. Exp. Neurol. 62(1):93-98.

D-9. Adult female mice exposed to 100 ppm H_2S for 2 h had reduced labelled leucine incorporation in cerebral protein and myelin 24 and 48 h after exposure. Incorporation returned to control level 72 h after exposure.

4-147 Evans, C. L. 1967. The Toxicity of Hydrogen Sulphide and Other Sulphides. Quart. J. Exp. Physiol. 52:231-48.

C-9. The injection of ~ 130 μmole/ml H_2S into rats and rabbits generally first increased then decreased respiration and blood pressure. Sometimes bradycardia appeared. H_2S appeared in the exhaled air. Rate and appearance of these responses varied with the site and rate of injection, suggesting detoxification in the blood. The action on nerve centers was reversible. Inhalation experiments (no data given) are supposed to have shown similar results.

4-334 Fischer, O., and E. Starkenstein. 1932. Chronic Occupational Hydrogen Sulfide Poisoning. Samml. Vergiftungsfällen. 3:27-31 (Ger).

C-9. Extensive data on one case of chronic H_2S poisoning of a lab worker preparing thiourea. Classic symptoms are described, and apparent increasing sensitivity with time.

4-302 Flury, F. 1928. Modern Tissue Poisons in Pharmacological-Toxicological View. Arch. Exp. Path. Pharmakol. 138:65-82.

D--. Review.

4-041 Foderman, V. M., N. S. Grishko, V. I. Rodin, A. M. Trankovski, and I. A. Nakrasova. 1979. Experimental Substantiation in Inhalation of Aerosols as Prophylaxis for Occupational Diseases of the Upper Respiratory Tract in Workers of Coal and Metal Mining Industries. Zh. Ushn. Nos. Gorl. Bolezn. (4):17-21 (Russ).

D--. H_2S in water (40 mg/L) was less effective than other prophylactics in delaying the development of experimentally induced silicosis and anthracosis in rats.

4-204 L. V. Fomicheva, and A. I. Gymrya. 1973. Eyes During Experimental Chronic Mercury Poisoning. Vestn. Oftal'mol. No. 2:77-80 (Russ).

D--. Changes in the eyes of rats induced by chronic mercury poisoning were improved by inhalation of H_2S aerosols.

4-336 Frankisek, M. V. 1950. Lesions of the Eye Due to Hydrogen Sulfide. Ceskoslovenka Oftal. 6:5-8 (Czech).

D--. Eye irritation was seen in workers in a sugar factory: blepharospasm, light sensitivity, and epithelial edema. Experimental studies with rats showed similar effects.

4-149 Freireich, A. W. 1946. Hydrogen Sulfide Poisoning--Report of Two Cases, One with Fatal Outcome, from Associated Mechanical Asphyxia. Am. J. Pathol. 22:147-155.

D-4. Results of the autopsy of the victim of sewer gas intoxication. No exposure data - time or concentration.

4-242 Fyn-Djui, D. 1961. See Duan Fyn-Zhui (1959).

4-205 Gaultier, M., J. Lecoeur, R. Le Breton, P. Gervais, and J. P. Fréjaville. Case of Extremely Acute Hydrogen Sulfide Poisoning with Symptoms of Pulmonary Involvement. J. Fr. Med. Chir. Thorac. 21(1):23-26 (Fre).

C-6. Brief description of 2 fatal and 3 light exposure cases. The fifth victim's symptoms included those described in most accidental H_2S exposure reports, plus abnormal electroencephalograms and slight jaundice. The same cases are described in Piva et al. (1974).

4-300 Glebova, L. F. 1960. Establishing Maximum Allowable Concentration of Hydrogen Sulfide. In: Atmospheric Air. B. S. Levine, Translator. U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. 3:98-101.

B-8. Breast-fed babies whose mothers were employed in a viscose spinning shop were exposed to 0.028-0.055 mg H_2S/m^3 (no CS_2) from the mothers' clothing. They were anemic, poorly developed, and susceptible to frequent and severe infectious diseases.

4-044 Gofmekler, V. A., I. I. Brekhman, V. G. Golotin, A. A. Sheparev, E. B. Krivelevich, L. N. Kamynina, A. I. Dobryakova, and V. A. Gonenko. 1977. The Embryotropic Action of Nitrogen Dioxide and a Complex of Atmospheric Pollutants. Gig. Sanit. No. 12:22-27 (Russ).

D--. An increased percentage of intrauterine destruction of embryos, various types of hemorrhages, and other developmental anomalies were seen in rabbits exposed to an atmosphere containing NO_2 or a mixture of atmospheric pollutants in which CO, phenol, and H_2S concentrations exceeded their maximum allowable concentrations.

4-206 Golyakova, L. P. 1971. Present-Day State of Industrial Hygiene Problems in Hydrometallurgical Production of Tungsten and Molybdenum Salts and Oxides. PB254580T, National Technical Information Service, U.S. Dept. of Commerce, Springfield, Virginia, 10 pp.

D-6. Dust, NH_3, H_2S, and HCl were encountered. H_2S concentrations were as high as 21-124 mg/m^3. No real exposure or toxicity data.

4-045 Goryacheva, I. G. 1979. Mathematical Experimental Planning in the Study of the Effect on a Living Organism of Several Harmful Elements Found in the Air. Khim. Tekhnol. (Kiev) No. 2: 50-54 (Russ).

D--. Typical factorial design. Use of regression analysis to evaluate the effect of CO, H_2S, CS_2, and CO, H_2S, and SO_2 on an organism.

4-259 Grant, W. M. 1962. Toxicology of the Eye. Charles C. Thomas, Pubisher, Springfield, Illinois. pp. 271-274.

D--. A review of ocular symptoms caused by H_2S, which generally start after several hours' exposure and include irritation, tearing, and burning. May increase to photophobia, colored haloes around lights, redness and swelling of lids, and fine gray stippling of the corneal epithelium. 100 ppm H_2S can cause immediate effects, but most H_2S keratoconjunctivitis caused by much lower concentrations.

4-283 Gurinov, B. P. 1952. Limits of Allowable Concentrations of Hydrogen Sulfide in the Atmospheric Air of Inhabited Localities. In: Limits of Allowable Concentrations of Atmospheric Pollutants. Book 1. V. A. Ryazanov, Ed. B. S. Levine, Transl. Office of Tech. Services, U.S. Department of Commerce, Washington, D.C. pp. 46-51.

A--. Recommendations for community H_2S standards of 0.05 mg/m^3 for a one-time limit and 0.015 mg/m^3 for a maximum avg. concentration. A review of Russian studies on atmospheric H_2S levels near various industries (0.01-1.25 mg/m^3) and toxic effects, many investigators maintaining that chronic H_2S poisoning will follow prolonged continuous exposure to extremely low concentrations of H_2S. Inhabitants exposed to tenths and hundredths of mg/m^3 complained of general debility, vertigo, nausea, and headaches.

4-296 Haggard, H. W. 1924. Action of Irritant Gases upon the Respiratory Tract. J. Ind. Hyg. 5(10):390-398.

D--. A general review. No specific mention of H_2S.

4-236 Haggard, H. W. 1925. The Toxicology of Hydrogen Sulfide. J. Ind. Hyg. Toxicol. 7:113-121.

D--. Review of older data, mostly high dose, in near lethal to lethal concentrations.

4-243 Haggard, H. W., Y. Henderson, and T. J. Charlton. 1922. The Influence of Hydrogen Sulfide upon Respiration. Am. J. Physiol. 61:289-297.

D-6. Experiments on single dogs. Exposure for 18 min to 1,000 ppm H_2S caused respiratory difficulty with apparent recovery. Death occurred at 2,000 ppm H_2S in 16 min, and in 34 min in a vagotomized dog.

4-234 Haider, S. S., M. Hasan, and P. Islam. 1980. Effect of the Air Pollutant Hydrogen Sulfide on the Levels of Total Lipids, Phospholipids, and Cholesterol in Different Regions of the Guinea Pig Brain. Indian J. Exp. Biol. 18(4):418-420.

D-11. Guinea pigs exposed to 20 ppm H_2S for 1 h/d for 11 d showed decreased lipid and phospholipid levels in some areas of the brain. Authors believe this to be due to increased peroxidation of endogenous lipids. A mechanistic study, with questionable applicability to this report. What is the significance of these changes?

4-150 Hand, B. M. 1939. Carbon Disulfide and Hydrogen Sulfide Hazards in the Viscose Rayon Industry. Hahnemannian Mon. 74:117-125.

D--. A review of the effects of CS_2.

4-047 Hays, F. L. 1972. Studies of the Effects of Atmospheric Hydrogen Sulfide in Animals. No. 73-21, 783, University Microfilms, Ann Arbor, Michigan. 217 pp.

B-13. Mice showed some signs of stress after 24 h exposure to 10 or 20 ppm H_2S, with at least partial recovery within several days. Goats and lactating dairy cows were generally unaffected until exposed to 50 ppm (exposure times from 24 h to 3 wk). Includes a review of effects and mechanism.

4-208 Hayes, F. L., E. Goret, H. D. Johnson, and L. Hahn. 1972. Hydrogen Sulfide Exposure in Ruminants. J. Anim. Sci. 35(1):189.

C--. An abstract of work completely presented in Hays (1972) [4-047].

4-325 Hays, F. L., and H. D. Johnson. 1972. Effect of Atmospheric Hydrogen Sulfide (H_2S) on Mice. Trans. Mo. Acad. Sci. 6:176.

C--. An abstract of work completely presented in Hays (1972) [4-047].

4-314 Henderson, Y., and H. Haggard. 1943. Noxious Gases and the Prin-
4-295 ciples of Respiration Influencing their Action. 2nd ed. Reinhold Publishing Corporation, New York, New York. pp. 140-141, 243-245.

D--. A short review of H_2S toxicity and mechanisms.

4-048 Henkin, R. I. 1976. Effects of Vapor Phase Pollutants on Nervous System and Sensory Function. In: Chemical Implications of Air Pollution Research. A. J. Finkel and W. C. Duel, Eds. Publishing Sciences Group, Acton, Massachusetts. pp. 193-216.

D--. Brief reviews of the toxicity of several gases, including H_2S.

4-049 Higuchi, Y. 1977. Behavorial Studies on Toxicity of Hydrogen Sulfide by Means of Conditioned Avoidance Responses in Rats. Folia Pharmacol. Jap. 73(3):307-320 (Japan).

D--. Discriminated avoidance response and Sidman-type avoidance response were inhibited by 200 and 300 ppm H_2S respectively, at the minimum. There was recovery in 1 h after exposure to low levels, but persistance of inhibition for a day after exposure to higher H_2S levels.

4-292 Holasova, P. 1969. Pathology of Children in an Area Polluted with Carbon Disulfide and Hydrogen Sulfide in Comparison with a Control Group. Cesk. Hyg. 14(7-8):260; Chem. Abstr. 72:35459v (1970).

D--. Children living near an industrial plant emitting 32 kg/h H_2S and 55.5 kg/h CS_2 had a 10% higher incidence of upper respiratory tract diseases than control children living in a rural area.

4-052 Horton, A. D., and M. R. Cuerin. 1974. Quantitative Determination of Sulfur Compounds in the Gas Phase of Cigarette Smoke. J. Chromatogr. 90(1):63-70.

C--. H_2S levels in cigarettes: 81 µg/cig (nonfilter), 89 µg/cig (cellulose acetate filter), 25 µg/cig (charcoal filtered), and 112 µg/cig (filtered little cigar).

4-152 Howes, H. S. 1944. Eye Inflammation as the Only Symptom of Incipient Hydrogen Sulphide Poisoning. Analyst 69:92.

D-4. Eye trouble in a tannery was attributed to H_2S. No real data were given.

4-291 Hromadka, M. 1965. Fatal Intoxication with Hydrogen Disulfide [Hydrogen Sulfide and Carbon Disulfide] in a Spinning Tank. Pracouni Lekar. 17(2): 68-69; Chem. Abstr. 62:16872e (1965).

D--. Description of a death occurring in an atmosphere of 6.1 mg H_2S/L and 3.8 mg CS_2/L, in the manufacture of viscose cords.

4-244 Hurwitz, L. J., and G. I. Taylor. 1954. Poisoning by Sewer Gas with Unusual Sequelae. Lancet 1:1110-1112.

D-5. Case report of severe, but nonfatal, intoxication due to a 30-min exposure to sewer gas, probably containing a high level of H_2S.

4-053 Husain, M. M. 1976. In Vitro Effect of Hydrogen Sulfide on the Activity of Some Enzymes of Rat Lung Homogenate. Ind. Health 14(3-4):93-96.

D-6. H_2S concentrations of 18.2-502 ppm in the homogenate caused changes in the activity of 6/7 rat lung enzymes, even at the lowest concentration. Pattern and degree of changes varied with the enzyme. Includes the same results reported in Husain and Zaidi (1977) plus two additional enzymes.

4-054 Husain, M. M., and S. H. Zaidi. 1977. An in Vitro Study on the Interaction of Hydrogen Sulfide with Enzymes of Rat Lung. In: Proceedings of the 1st International Symposium of Environmental Pollution and Human Health. pp. 458-464.

D-11. Concentrations of 18.2-502 ppm H_2S were bubbled through rat lung homogenates, inhibiting acid phosphatase, alkaline phosphatase, and glutamic-pyruvic transaminase, even at 18 ppm. Arginase activity increased with increased H_2S concentration. The authors suggested that these changes could lead to the deprivation of amino acids needed for protein biosynthesis in lung tissue.

4-055 Hysing, E. S., and E. Wergeland. 1975. Gas Exposure and Health Problems in Herring Oil and Fish Flour Industry. Tidsskr Nor. Laegeforen. 95(4):226-228 (Nor).

C--. Review of toxic gases to which employees in the herring oil and fish flour industry are exposed. Also includes a case report. The autopsy revealed the typical results of H_2S poisoning.

1-0172 ILO. 1970. Permissible Levels of Toxic Substances in the Working Environment. Occupational Safety and Health Series 20, International Labour Office, Geneva. pp. 194-198.

C--. Maximum Acceptable Concentrations in Czechoslovakia.

	Normal MAC (mg/m^3)	Short, Single Exposure MAC (mg/m^3)
Acrolein	0.5	1.0
NH_3	40	80
HCHO	2	5
HCN	3	15
MeOH	100	500
H_2S	30	-

4-153 ILO/WHO, International Labour Office/World Health Organization, Committee on Occupational Health. 1970. Permissible Levels of Toxic Substances in the Working Environment. In: Proceedings of the Sixth Session of the Joint ILO/WHO Committee on Occupational Health, Geneva, Switzerland. June 4-10, 1968. International Labour Office, Geneva, Switzerland. pp. 190, 195, 202, 218, 235, 242, and 334.

D--. The Maximum Acceptable Concentration (MAC) for H_2S in several countries ranges from 10 to 30 mg/m^3.

4-056 Ivanenko, N. A. 1976. Clinical Aspects and Treatment of Severe Hydrogen Sulfide Poisoning. Vrach. Delo. No. 8:126-128 (Russ).

C-6. An extensive description of two victims of sewer gas poisoning. No H_2S concentration data. Complications and remote sequelae didn't occur. Used cyanide-type treatment.

4-318 Jaensch, P. A. 1930. Symptomology and Therapy of Eye Injuries Caused by Hydrogen Sulfide. Arch. Gewerbepathol. Gewerbehyg. 1:397-407 (Ger).

D--. Review of clinical aspects of H_2S exposure with regard to the eye. Jaensch recommends that work room air contain $\leq$500 ppm to avoid eye injury and preferably much less.

4-306 Jonek, J., and J. Konecki. 1966. Histochemical Examination of Pulmonary Enzyme Level Changes in Experimental Hydrogen Sulfide Poisoning. Med. Pracy. 17(4):329-335 (Pol).

C--. The exposure of rabbits to 0.1 mg H_2S/L (this is the best guess of the dose from the unclear photocopy we received of the article) for 0.5 h/d for 7, 10, or 14 d caused decreased activity of 4 enzymes in the lung.

4-057 Jones, J. P. 1975. Hazards of Hydrogen Sulfide Gas. Sel. Pap. Annu. Gas Meas. Inst. 23rd, 16. 4 pp.

D--. A general discussion of the dangers of H_2S to humans. Some workplace precautionary measures are given.

4-154 Kaipainen, W. J. 1954. Hydrogen Sulfide Intoxication--Rapidly Transient Changes in the Electrocardiogram Suggestive of Myocardial Infarction. Ann. Med. Intern. Fenniae. 43:97-101.

D-4. Case report of probable H_2S intoxication contracted in shovelling manure, of unknown strength and duration. Unconsciousness and convulsions resulted. Changes in the electrocardiogram resembling a myocardial infarction lasted several days.

4-209 Kami´nski, M., and P. Mikolajczyk. 1967. Changes of the Activity and Localization of Acid Phosphatase, Alkaline Phosphatase and Desoxyribonuclease II in Motoric Cells of the Anterior Horn of the Spinal Cord in Rabbits in Acute Hydrogen Sulfide Poisoning. Med. Pr. 18(1):42-46 (Pol).

C--. Rabbits exposed to 100 mg/m^3 for 0.5 h/d for 7, 10, or 14 d were decapitated at the end of the exposure periods, and tissue sections were examined.

4-322 Kamnev, V. M., and T. M. Lyutikova. 1965. The Action of a Mixture of Hydrocarbons and Hydrogen Sulfide on the Histological Structure of the Central Nervous System. Tr. Omsk. Med. Inst. 61:141-146 (Russ).

D--. Microscopic changes in the brains of mice exposed to the title mixture are described. The concentrations of the components in the mixture to which the mice were exposed and of the total mixture itself were not given. The mixture was generated by a thermal cracking unit.

4-060 Kaplun, S. Ya., and E. G. Koptyeva. 1973. Age-Related Characteristics of Reactions to Hydrogen Sulfide by its Indices in Blood and Changes of Arterial Pressure and Respiration. Fiziol. Zh. (Kiev) 19(3):328-332 (Ukr).

D--. Dogs were dosed i.v. by H_2S.

4-059 Kaplun, S. Ya., E. G. Kopteva, E. E. Grechishcheva, and L. K. Briukhanova. 1975. Development of Experimental Myocarditis under Conditions Using Various Concentrations of Hydrogen Sulfide Baths. Vopr. Kurortol. Fizioter. Lech. Fiz. Kult. No. 1:56-60 (Russ).

C--. Fewer rats and rabbits succumbed to experimentally induced myocarditis if they were subsequently given H_2S baths containing 150 or 300 mg H_2S/L. The resistance of their erythrocytes to hemolysis decreased under the influence of the H_2S baths.

4-061 Kappus, H. 1979. Toxicology of Hydrogen Sulfide. Staub - Reinhalt. Luft. 39(5):153-155 (Ger).

D--. A review with 6 references.

4-155 Kemper, F. D. 1966. A Near-Fatal Case of Hydrogen Sulfide Poisoning. Can. Med. Assoc. J. 94:1130-1131.

D-4. Duration of exposure and the concentration of the gas are unknown. A detailed description of the treatment is given, including hypothermia.

4-063 Khusainov, Z. K., and A. N. Titov. 1977. Hygienic Evaluation of the Air in Pig-Sties of the Large-Scale Farming Type. Vestn. S-kh. Nauki Kaz. 20(5):75-78 (Russ).

D--. Pig-sty air contained 15.0-22.5 mg NH_3/m^3, 0.055-0.102 vol. % CO_2, and 1.26-1.50 mg H_2S/m^3.

4-065 (2-0018) Kiryakov, K., T. S. Vodichenska, V. Markovska, and E. Tsutsulova. 1978. Contamination of the Atmospheric Air and Morbidity with Temporary Loss of Working Capacity Among Agrarian Workers. Khig. Zdraveopaz. 21(1):42-48 (Bulg).

D-5. An epidemiological study of 2 similar groups of people showed an increase in general morbidity and in specific groups of diseases (respiratory and neuritis) in the group exposed to a mixture of H_2S, phenol, and HCN in the atmosphere.

4-156 Kleinfeld, M. 1965. Acute Pulmonary Edema of Chemical Origin. Arch. Environ. Health 10:942-946.

D-5. Only a brief mention of H_2S. A 15-min exposure to unknown level of H_2S caused pulmonary edema, clearing in 24 h.

4-157 Kleinfeld, M., C. Giel, and A. Rosso. 1964. Acute Hydrogen Sulfide Intoxication--An Unusual Source of Exposure. Ind. Med. Surg. 33:656-660.

C-5. Several case reports from the accidental exposure of 89 people to unknown, but high, levels of H_2S. Dizziness, nausea, pulmonary edema, apnea, unconsciousness, respiratory arrest, and death were seen.

4-066 Klentz, R. D., and M. R. Fedde. 1978. Hydrogen Sulfide: Effects on Avian Respiratory Control and Intrapulmonary CO_2 Receptors. Respir. Physiol. 32(3):355-368.

B-12. Acute exposure of chickens to high levels of H_2S caused changes in lung function and intrapulmonary CO_2 receptors, and death at 4,000 ppm.

4-284 Kononova, V. A., and V. B. Aksenova. 1961. Contamination of the Atmosphere by Emissions from a Plant Producing Synthetic Alcohol and their Effect on the Morbidity and Sanitary-Domestic Life of the Population. Gig. Sanit. 26:3-7 (Russ).

D--. The plant contaminated its environs with SO_2, H_2S, and hydrocarbons up to a distance of 3 km and with unsaturated hydrocarbons for a distance of 500 m. The disease incidence for children living near the plant in 1957-1959 was greater than that for children of the control group.

4-067 Koptyeva, E. G., S. Ya. Kaplun, and L. K. Bryukhanova. 1973. Absorption Properties of Tissues with Experimental Myocardial Dystrophy. Fiziol. Zh. (Kiev) 19(6):824-829 (Ukr).

C--. The author abstract of this Ukrainian article states that the effects of H_2S baths, taken before drug-induced myocardial dystrophy, on the metabolic processes and resistivity of tissues depended on the H_2S concentration in the baths. The baths contained 150 or >300 mg H_2S/L.

4-298 Kosmider, S., and E. Rogala. 1967a. Electrolyte Balance Disturbances in Serum and Tissues in Subacute Experimental Poisoning with Hydrogen Sulfide. Int. Arch. Gewerbepathol. Gewerbehyg. 23:12-18 (Ger).

C-10. Rabbits exposed to 100 mg H_2S/m^3 for 1 h/d for 5 d showed electrolyte disturbances in the blood serum and tissues (especially the brain) and heart rhythm disturbances. P, Fe and Cu values were high in the tissues (liver and brain), but not in the serum.

4-211 Kosmider, S., and E. Rogala. 1967b. Mineral Metabolism Disorders in Blood Serum and Tissues in Experimental Subacute Hydrogen Sulfide Poisoning. Int. Arch. Arbeitsmed. 23(1):12-18 (Ger).

C-8. Rabbits were exposed to 100 mg H_2S/m^3 for 1 h/d for 5 d. Significant changes were observed relative to the controls in the concentrations of P and Na in the blood serum; Na, P, Fe, and Cu in brain homogenate; and Fe in liver homogenate.

4-212 Kosmider, S., and K. Zajusz. 1966. Various Enzyme and Mineral Metabolism Disorders in the Cerebellum in Experimental Subacute Hydrogen Sulfide Poisoning. Zentrabl. Allg. Pathol. Anat. 109(4):411-417 (Ger).

C-8. Rabbits were exposed to 100 mg H_2S/m^3 for 1 h/d for 7 d, which disturbed the concentrations of Na and Fe and the activity of alkaline phosphatase in the brain compared to those of the controls.

4-213 Kosmider, S., E. Rogala, and A. Pacholek. 1966. Studies on the Toxic Action Mechanism of Hydrogen Sulfide. Int. Arch. Gewerbepathol. Gewerbehyg. 22(1):60-76 (Ger).

C-9. Rabbits were exposed to 100 mg H_2S/m^3 for 1.5 h or for 0.5 h/d for 5 d, which caused enzyme, mineral, protein, and EKG disturbances. Inhibition of the activity of aerobic enzymes and of those concerned with active transport in the cerebellum and heart may depend causally on the functional disturbances of these organs. The decrease of alkaline phosphatase activity is traceable to the reaction of H_2S with the enzymatically active Mg ions, and the Na citrate-reversible heart-rhythm disturbances observed in the subacute poisoning are attributed to the increase of biologically active Ca ions within the heart muscle.

4-158 Kosmider, S., E. Rogala, and A. Pacholek. 1967. Electrocardiographic and Histochemical Studies of the Heart Muscle in Acute Experimental Hydrogen Sulfide Poisoning. Arch. Immunol. Ther. Exp. 15:731-740.

C-9. Both acute (1.5 h) and repeated (0.5 h/d, 5 d) exposure of rabbits to 100 mg H_2S/m^3 caused abnormal EKG's, though of different types. Enzyme activity in myocardial cells decreased, and possible calcium disorder was seen.

4-159 Kosmider, S., E. Rogala, J. Dwornicki, and Z. Szulik. 1971. The Influence of Vitaral on some Mineral and Enzyme Disturbances in Subacute Poisoning with Hydrogen Sulfide. Int. Arch. Arbeitsmed. 29:64-84.

C-10. Rabbits exposed to 100 mg H_2S/m^3 1 h/d for 14 d had protein, mineral, and enzymatic disturbances in the blood, liver, kidney, and brain. These were normalized by the repeated ingestion, during H_2S exposure, of a vitamin and mineral supplement.

4-068 Kosmider, S., E. Rogala, Z. Szulik, and J. Dwornicki. 1973. Effect of Trace Metals on Disorders of Acid-Base Equilibrium and Mineral Metabolism in Subacute Poisoning with Hydrogen Sulfide. Patol. Pol. 24(1):171-186 (Pol).

C--. Exposing rabbits to 100 mg H_2S/m^3 for 1 h/d for 14 d caused mineral and acid-base balance disorders in the blood, which disturbances were improved by Vitaral (vitamin-mineral preparation) treatment in the course of the poisoning.

4-069 Kosmodamianskaya, D. M., and S. F. Sorokina. 1976. Characteristics of Allergic Pathologies of the Child Population in an Industrial Center. Gig. Sanit. No. 11:101-102 (Russ).

D--. Children in an industrial center who were exposed to numerous chemicals (including CO, H_2S, SO_2, and NH_3) in concentrations often exceeding maximum permissible levels showed a 1.5-3 times greater incidence of respiratory afflictions and 10 times greater incidence of allergic dermatitis than for children living in an area where the chemicals were below acceptable limits.

4-070 Kostlan, J. 1976. Contribution to the Analysis of the Possibility of Escape of Workers during Accidental Emission of Gases. Sb. Pr. Vyzk. Chem. Vyuziti Uhli, Dehtu Ropy. 14:253-264 (Czech).

D--. The maximum atmospheric concentration of H_2S through which a man might run with restricted breathing, with the assumption that the worker can reach a safe area within 1 min, is 1,000 ppm.

4-160 Kranenberg, W. R. H., and H. Kessener. 1925. Hydrogen Sulfide and Carbon Disulfide Poisoning. Zentralbl. Gewerbehyg. Unfallverhuet. 2:348-350 (Ger).

B-9. Workers in a sugar beet processing plant, exposed to ~14-37 mg H_2S/m^3, had repeated eye irritations. Includes a review of the viscose rayon industry, with workers exposed to H_2S (~9-86 mg/m^3), CS_2, and H_2SO_4. The acid may have played a role in the eye problems that occurred.

4-214 Krasovitskaya, M. L., T. A. Shirinkina, S. G. Zinov'eva, and T. N. Novoselova. 1970. A Sanitary Evaluation of a Chemical Combine as the Source of Contamination of the Atmospheric Air. Tr. Permsk. Med. Inst. 82:40-45 (Russ).

D--. Children living in an area polluted by H_2S, SO_2, N oxides, phenol, H_2SO_4, and dust showed a lag in physical development, elevation in incidence of anemia and chronic rhinitis, and more frequent central nervous system disturbances than controls.

4-286 Krekel, K. 1964. Electrocardiographic (ECG) Changes in Two Workers after Hydrogen Sulfide Poisoning. Zentralbl. Arbeitsmed. Arbeitsschutz. 14(7):159-163 (Ger).

C-6. Two men were briefly exposed to an unknown (probably low) level of H_2S. Extensive physical exam found no real problems except for some EKG abnormalities and moderate dyspnea. One complained of continuing dizziness and fatigue, but had a complicating head injury so symptoms cannot be attributed only to H_2S.

4-071 Kriz, J., L. Pelech, Z. Madlo, and D. Wokounova. 1976. Bone Age in Children from Areas with Polluted Atmospheres. Cesk. Hyg. 21(6-7):326-330 (Czech).

D--. Bone maturation and serum alkaline phosphatase were reduced in two groups of 9-y-old children exposed to various chemicals. One group was exposed to H_2S, CS_2, SO_2, Cl_2, and H_2SO_4 in the ambient air; the other, to SO_2, dust, and emissions from a synthetic rubber manufacturing plant.

4-161 Kuwai, S. 1960. Experimental Studies on Gas Inhalation of Respective and Combined H_2S. Shikoku Igaku Zasshi. 16:144-164 (Japan).

B-10. Includes an English abstract. Nice study, including interaction. Exposing rabbits to 20-25 ppm H_2S for 4 h/d for 150 d caused no change in general state or body weight, and a temporary increase in free cholesterol. Combined inhalation with 20-25 ppm CS_2 caused more symptoms than either gas alone.

4-073 Langner, R. R., S. K. Norwood, G. E. Socha, and H. R. Hoyle. 1979. Two Methods for Establishing Industrial Hygiene Priorities. Am. Ind. Hyg. Assoc. J. 40(12):1039-1045.

D--. A theoretical discussion, with H_2S as one of several compounds briefly used for demonstration.

4-237 Larsen, V. 1944. Eye Disease Caused by Hydrogen Sulfide in Tunnel Workers. Acta Ophthalmol. 21:271-286 (Fre).

A-9. Eye irritation was seen in workers exposed to ~40-185 mg H_2S/m^3.

4-245 Larson, C. P., C. C. Reberger, and M. J. Wicks. 1964. The Purple Brain Death. Med. Sci. Law 4:200-202.

D-4. Case report of a fatality in a plant making fertilizer from chicken feathers. The death is attributed to H_2S poisoning, and the brain color to sulphaemoglobin. A more detailed description is given in Breysse, 1961.

4-162 Laug, E. P., and J. H. Draize. 1942. The Percutaneous Absorption of Ammonium Hydrogen Sulfide and Hydrogen Sulfide. J. Pharmacol. Exp. Therap. 76:179-188.

D-8. No inhalation exposure. H_2S exposure of rabbits was through the skin, at unknown concentrations, and death was the endpoint measured.

4-311 Layton, D., Ed. 1980. An Assessment of Geothermal Development in the Imperial Valley of California, Volume 1-Environment, Health and Socioeconomics. DOE/EV-0092, National Technical Information Service, U.S. Department of Commerce, Springfield, Virginia. pp. 10-3 to 10-9.

D--. Includes a 7-page review of public health considerations (particularly odor theshold) of H_2S, as the major health problem related to thermal energy utilization.

4-215 Lebedeva, E. K., and M. I. Skorospeshkina. 1970. The Morbidity Among Children of the Town of Perm Living Near Chemical Factories. Tr. Permsk. Med. Inst. 82:73-76 (Russ).

D--. A group of 1,000 infants (<1 y) living in an area with several sources of atmospheric pollutants (dust, carbon black, SO_2, H_2S, HF, SO_3, NO_x, and hydrocarbons) was compared to a control group of 1,000. There was more respiratory illness (pneumonia, bronchitis) and exudative diathesis in the exposed group.

4-216 Leduc, G. 1967. Report of a Study of the Pathological Consequences of Chronic Exposure to Sulfur Dioxide, Hydrogen Sulfide, and Chlorine Dioxide. Arch. Mal. Prof. 28(1):307-311 (Fre).

C-5. A review of several industries in France, stating that most H_2S levels encountered had no effect, but didn't give any concentration information. During "bad periods," workers complained of headaches, epistases, itching, and digestive troubles (vomiting, constipation, diarrhea).

4-163 Legge, T. M. 1934. Industrial Maladies. Oxford University Press, London. pp. 146-151.

D--. Reminiscences of many years of experience. Eye inflammation was the major symptom.

4-238 Lehmann, K. B. 1892. Experimental Studies on the Habituation to Industrial Gases. VI. Hydrogen Sulfide. Arch. Hyg. 14:135-189 (Ger).

B-9. The original studies. Also reviews much work done earlier. Cats, rabbits, and dogs were exposed to very high levels (1,420-48,280 mg/m^3) for up to several hours. The lower levels caused minor mucous membrane irritation, and the higher levels caused convulsions. Most recovered. Human exposure ranged from 284 to 8,165 mg/m^3, with no apparent symptoms at the lowest level.

5-149 Leonardos, G., D. Kendall, and N. Barnard. 1969. Odor Threshold Determinations of 53 Odorant Chemicals. J. Air Pollut. Control Assoc. 19(2):91-95. Data also appear in A.D. Little, Inc., Research on Chemical Odors, Manufacturing Chemists Assoc., Washington, D.C. 1968.

A-11. Definitive paper. The odor recognition threshold for H_2S was 0.00047 ppm, and 0.0047 ppm when H_2S was derived from Na_2S.

4-299 Lewey, F. H. 1938. The Health Hazards in the Viscose Rayon Industry. In: Pennsylvania Dept. of Labor and Industry. Survey of Carbon Disulfide and Hydrogen Sulphide Hazards in the Viscose Rayon Industry. Bull. 46, Occupational Disease Prevention Division, Pennsylvania Dept. of Labor and Industry, Harrisburg, Pennsylvania. pp. 17-22.

D--. A review of the occurrence and toxicity of CS_2. A brief mention of H_2S, that workers exposed to 16-25 ppm H_2S had conjunctivitis and those exposed to 12-16 ppm had no complaints.

4-074 Lewicki, L., and W. Turkiewicz. 1979. Problems of the Maximum Permissible Concentrations of Toxic Substances in Air. Cuprum. 6(1):32-36 (Pol).

D--. A discussion of the problems of determining the maximum permissible concentrations of CH_4, CO_2, NO_2, SO_2, Hg, and H_2S in the air of mines.

4-316 Loginova, R. A. 1957. Basic Principles for the Determination of Limits of Allowable Concentrations of H_2S in Atmospheric Air. In: Limits of Allowable Concentrations of Atmospheric Pollutants III, V. A. Riazanov, Ed. National Technical Information Service, U.S. Department of Commerce, Springfield, Virginia. pp. 52-68.

A-10. An extensive review of Russian work on the toxicity of H_2S to humans, as well as reports of 2 new studies. The odor threshold is given as ~0.04 mg/m^3. H_2S air pollution in the presence of hydrocarbons is considered to pose a greater danger than H_2S alone. Authors recommend 0.03 mg/m^3 as the limit of allowable concentrations for pure H_2S, and 0.015 mg/m^3 for H_2S as a constituent of natural gas.

4-344 Ludvich, D. 1981. The Hydrogen Sulfide Technical Manual. Safety Technology and Oilfield Protectors, Inc. Lafayette, Louisiana. 123 pp.

D--. A safety and handling training manual.

4-164 Lund, O. E., and H. Wieland. 1966. Pathologic-Anatomic Findings in Experimental Hydrogen Sulfide Poisoning (H_2S)--A Study on Rhesus Monkeys. Int. Arch. Gewerbepathol. Gewerbehyg. 22:46-54 (Ger).

B-8. Acute exposure ($\leqq$35 min) of monkeys to 500 ppm H_2S caused frequent rubbing of the eyes (mucous membrane irritation), gasping, and unconsciousness in ~15 min. One died immediately, one 5 d later, and one survived. Autopsies were done on all 3, emphasizing the brain.

4-217 Lutsenko, L. A. 1970. Industrial Hygiene in the Concentration of Sulfide Copper Ores at Mills in the Urals. Gig. Tr. Prof. Zabol. 14(10):11-15 (Russ).

D--. Any health effects observed in this study would be confounded by all the possible chemical and physical exposures at an ore beneficiation plant. H_2S, HCN, and CS_2, for example, are decomposition products of the flotation reagents.

4-218 Manz, A. 1968a. On the Behavior of Tissue Oxidase and the Effect of Oxygen Administration in Hydrogen Sulfide Poisoning in the Animal Experiment. Zentralbl. Arbeitsmed. 18(11):325-333 (Ger).

D--. Mechanism study. Rabbits and/or guinea pigs were exposed to 1,500 or 1,900 ppm H_2S for $\leqq 210$ s and also to 5 and 20% H_2S.

4-219 Manz, A. 1968b. On a Case of Survival after Acute Poisoning by a Mixture of Hydrogen Sulfide and Hydrocyanic Acid. Zentralbl. Arbeitsmed. 18(6):167-171 (Ger).

D--. Cannot separate H_2S and HCN effects.

4-076 Matsuo, F., J. W. Cummins, and R. E. Anderson. 1979. Neurological Sequelae of Massive Hydrogen Sulfide Inhalation. Arch. Neurol. 36(7):451-452.

C-5. The results of a computerized tomography scan of the head of a victim of H_2S poisoning (level unknown, but high).

4-220 (2-0167) Mattina, C. F., Jr. 1972. A Potentiometric Method for the Determination of Hydrogen Cyanide and Hydrogen Sulfide in Cigarette Smoke. Tob. Sci. 16:113-114.

C--. Unfiltered cigarettes contained 4.3 μg H_2S/puff and 35.0 μg HCN/puff.

4-269 Mazanowski, A., and A. Mazanowska. 1964. Ventilation in Henstalls and Egg-Laying Performance. II. Mitt. Drobiarstwo, Warsaw. 12(11):10-11 (Pol).

D--. 15-75 mg H_2S/m^3 were reported in livestock quarters.

4-167 McCabe, L. C., and G. D. Clayton. 1952. Air Pollution by Hydrogen Sulfide in Poza Rica, Mexico. Arch. Ind. Hyg. Occup. Med. 6:199-213.

C-8. Extensive description of an accident hospitalizing 320 people because of exposure to unknown levels of H_2S. There was extensive irritation of the respiratory, gastrointestinal, and nervous systems. There were no respiratory or digestive sequelae.

4-168 McCormack, M. F. 1975. Sewer Fume Poisoning. J. Am. Coll. Emergency Physicians 4:141-142.

D-4. Case report of 2 acute H_2S poisonings, resulting in 1 death and 1 successfully treated victim. That man was unconscious, cyanotic, hyperventilating, and suffering from pulmonary edema and various central nervous system effects.

4-246 McDonald, R. 1938. Ophthalmological Aspects of CS_2 Intoxication. In: Pennsylvania Dept. of Labor and Industry. Survey of Carbon Disulfide and Hydrogen Sulphide Hazards in the Viscose Rayon Industry. Bull. 46, Occupational Disease Prevention Division, Pennsylvania Dept. of Labor and Industry, Harrisburg, Pennsylvania. pp. 38-40.

D--. Includes a short discussion of eye irritation due to H_2S. No concentrations given. Mentions large differences in individual susceptibility.

s4-169 McDougall, J. W. G., and T. O. Garland. 1954. Hydrogen Sulphide Gas Poisoning at Rotorua. N. Z. Med. J. 53:471-475.

D-6. Data on gases in a thermal springs area: 63% by vol. H_2S in one manhole. Descriptions of 3 fatalities, with H_2S implicated.

4-077 McQuitty, J. B., and J. R. Feddes. 1978. Warning: Manure Gases are Dangerous. Agric. For. Bull. 1(1):10-14.

D--. Short review of the occurrence and possible problems of several gases in barns, including NH_3 and H_2S.

4-247 Medvedev, V. M. 1959. The Effect of Certain Industrial Poisons on the Mechanism of the Nerve Impulse Transmission in Superior Cervical Sympathetic Ganglion. Report I. Acute Experiments with Hydrogen Sulfide, Ethylene and Propylene in Healthy Animals. Byull. Eksp. Biol. 47(4):79-82 (Russ).

D--. The upper cervical sympathetic ganglia of cats were perfused by an injected solution of H_2S in saline. The effect on the contraction of the nictitating membrane upon application of an electrical current was measured. H_2S lowered the humoral, not the electrical, transfer of the nerve impulse.

4-221 Mel'nichenko, R. K. 1968. Combined Effect of Carbon Monoxide and Hydrogen Sulfide. Vrach. Delo. 7:87-90 (Russ).

B-7. Rats were exposed to 300 mg H_2S/m^3 and 300 mg CO/m^3 or 300 mg CO/m^3 alone for 4 h/d for 6 mo. The study indicated that the action of the combined gases elicited a more expressed toxic effect than CO alone, in terms of the lowering of the O_2 absorption by the animals, inhibition of the phagocytic reactions, strengthening of the permeability of the vessels, and a tendency to the absence of a weight increment in the animals.

4-170 Milby, T. H. 1962. Hydrogen Sulfide Intoxication--Review of the Literature and Report of Unusual Accident Resulting in Two Cases of Nonfatal Poisoning. J. Occup. Med. 4:431-437.

C-6. A review of toxicity and treatment of H_2S inhalation. H_2S level estimated to have been around 2,000 ppm. Victims suffered loss of consciousness, convulsions, and no pulmonary edema. Rapid recovery.

4-222 Misiakiewicz, Z., G. Szulinska, and A. Chyba. 1972. Effect of a Carbon Disulfide-Hydrogen Sulfide Mixture in Air on White Rats during Several Months of Continuous Exposure. Rocz. Panstw. Zakl. Hig. 23(4):465-475 (Pol).

D-6. Rats exposed for 6 mo to 0.1 mg/m^3 each CS_2 and H_2S (combined) showed a 30% increase in blood cholinesterase activity, a 14% increase in coproporphyrin excretion in the urine, a 64% increase in aspartate aminotransferase serum activity, and bronchitis. The only change observed in rats exposed to 0.1 mg CS/m^3 for 6 mo was a 16% increase in blood cholinesterase.

4-239 Mitchell, C. W., and W. P. Yant. 1925. Correlation of the Data Obtained from Refinery Accidents with a Laboratory Study of Hydrogen Sulfide and its Treatment. Bull. No. 231. Bureau of Mines, U.S. Dept. of Commerce. pp. 59-80.

A-10. Review of human accidental/occupational exposure. A small amount of human experimental data. Definite irritation was seen at 100 ppm. Recovery was rapid in acute cases, and slower (days) in subacute cases. Acute exposure of canaries, rats, dogs, guinea pigs, and goats to a range of H_2S concentrations (35-1,600 ppm). Authors believe that man behaves similarly to cats and dogs in H_2S exposure.

4-223 Morie, G. P. 1971. Determination of Hydrogen Sulfide in Cigarette Smoke with a Sulfide Ion Electrode. Tob. Sci. 15:107.

C--. Unfiltered cigarettes contained 30-49 μg H_2S/cig. The amount was reduced by certain types of filters (18.7 μg/cig with acetate-carbon filter).

4-319 Moser, P. 1940. Effect of Prolonged Inhalation of Hydrogen Sulfide in Dogs. Arch. Exp. Pathol. Pharmakol. 196:446-454 (Ger).

B-11. Dogs exposed to 14 mg H_2S/m^3 for 7 h/d for 14 d showed only increased drinking. Exposure to ~150 mg/m^3 for 2 mo (7 h/d) caused some eye and respiratory problems, which cleared up after exposure stopped each day, and some slight blood changes.

4-081 Muller, P. H., L. Lenoir, D. Furon, M. Willot, A. Debarge, and J. P. deMendonca. 1973. Double Poisoning by Hydrogen Sulfide. Med. Lag. Domm. Corpor. (Paris). 6(3):284-286 (Fre).

C-8. Case report of 2 deaths attributed to H_2S in sewer gas, with intense tracheal congestion, alveolar and interstitial edema of the lungs, and congestion of the viscera.

4-082 Nabiullin, R. G., F. S. Khusainova, A. G. Zaerov, V. I. Nikulicheva, and N. M. Zagidullina. 1976. Role of Hydrogen Sulfide in the Development of Impairment of the Bronchopulmonary Apparatus in Petroleum Industry Workers. V Sb., Gig. Tr. Zabol. Neft. Neftekhim. Prom-st'. 74-77 (Russ).

D--. Petroleum workers, who are exposed to hydrocarbons as well as H_2S, showed more chronic bronchitis if they had worked ≧10 y compared to workers who had been exposed <10 y. The levels of H_2S and hydrocarbons as well as the numbers of workers examined were not given.

4-083 Nakajima, K., S. Tsuchiya, T. Nakajima, and K. Harada. 1975. The Effect of Nasal Obstruction on the Susceptibility of Mice to Noxious Gases (author's transl). Exp. Anim. (Tokyo). 24(2):45-52 (Japan).

D--. The threshold values for loss of righting reflex and respiratory arrest were higher for mice forced to breathe through the mouth (1,200 and 1,600 ppm) than for nasal breathing mice (1,000 ppm).

4-086 Natusch, D. F. S., and B. J. Slatt. 1978. Hydrogen Sulfide as an Air Pollutant. Air Pollut. Control 3:459-518.

B--. Review of natural and industrial sources of atmospheric H_2S, sampling and analytical methodologies, and corrosion of metals and paint. Reported odor threshold was 0.025-0.1 ppm normally, but lower when oil vapor is present, 0.006-0.02 ppm.

4-172 Nesswetha, W. 1969. Eye Lesions Caused by Sulphur Compounds. Arbeitsmed. Sozialmed. Arbeitshyg. 4:288-290 (Ger).

D--. Review.

6-124 Newsome, J. R., V. Norman, and V. L. Zaratzian. 1965. Vapor Phase Analysis of Cigarette Smoke. Tob. Sci. 9:102-110; or Tobacco 161(4):24-32.

D--. Levels in tobacco smoke (µg/40 mL puff):

	Unfiltered	Filtered
Methanol	13	10
HCHO	4.1	3.6
Acrolein	8.2	7.9
HCN	32	29
H_2S	3.4	3.1
NH_3	12	13

4-086 Nikolov, S. K., N. A. Kambulin, and M. V. Kolupaeva. 1976. Problems of Labor Hygiene in Modern Stock Breeding. Gig. Sanit. No. 12:80-84 (Russ).

D--. Review. Various authors have found up to 39 mg H_2S/m^3 and 3-135 mg NH_3/m^3 in large cattle, swine, and poultry-breeding establishments.

4-084 NRC, National Research Council, Subcommittee on Hydrogen Sulfide. 1979. Medical and Biological Effects of Environmental Pollutants. Hydrogen Sulfide. University Park Press. Baltimore, Maryland. 172 pp.

A--. Authoritative and extensive review of the effects of H_2S.

4-088 Odera, G. M. 1975. Fatality Produced by Accidental Inhalation of Drain Cleaner Fumes. Clin. Toxicol. 8(5):547-551.

D-4. Grossly incomplete data for the purpose of this study. Just a description of the accident, and a discussion of the safety of drain cleaners.

4-270 O'Donoghue, J. G. 1961. Hydrogen Sulphide Poisoning in Swine. Can. J. Comp. Med. Vet. Sci. 25:217-219.

C-8. The acute exposure of a few pigs and rabbits to increasing H_2S concentrations (50-1,200 ppm) caused death at longer exposures to the highest concentrations, and symptoms with apparent recovery after brief high exposures.

4-249 Oliver, T. 1911. The Sulphur Miners of Sicily: Their Work, Diseases, and Accident Insurance. Br. Med. J. 2:12-14.

D-4. Just a mention of the presence of H_2S in the mines and of a fatal accident claiming 11 lives.

4-233 Ostrow, D. N., J. Manfreda, K. S. Tse, T. Dorman, and R. M. Cherniack. 1978. Alpha_1-Antitrypsin Phenotypes and Lung Function in a Moderately Polluted Northern Ontario Community. Can. Med. Assoc. J. 118(6): 669-672.

D-8. Just mentions the presence of H_2S, at levels >20 ppm. Any abnormalities in the tested lung functions seem more likely to be due to dust exposure, which was also heavy. No comparisons with nonpolluted areas. The aim was to find differences in lung function due to phenotypes, not air quality.

4-271 Petkov, G. 1964. A Study of the Microclimate in Large Poultry Houses. Veterinarnomed. Nauki. Sofia 1(4):81-85 (Bulg).

D--. The levels of NH_3, H_2S, and CO_2 in poultry houses were above acceptable levels, and caused decreased hemoglobin and erythrocytes in the chickens.

4-175 Petrun, N. M. 1965. Indications of Poisoning by Hydrogen Sulfide Entering the Body Through the Skin. Farmakol. Toksikol. 28:488-490 (Russ).

C-9. Rabbits whose skin (except that of the head) was exposed to air containing 1,000 or 2,000 mg/m^3 showed inhibition of blood carboanhydrase and cholinesterase activities, reduced erythrocyte respiration rate, changes in the gaseous composition of the blood, and reduced hemoglobin.

4-176 Pettigrew, G. L. 1976. Preliminary Report on Hydrogen Sulfide Exposure in the Oil and Gas Industry. Public Health Service, Dallas Regional Office, U.S. Dept. of Health, Education, and Welfare. 4 pp.

D--. A brief review of the occurrence of H_2S poisoning, and suggestions for reducing the hazard. No concentration or effects information given.

4-090 Piva, C., O. Diamant-Berger, J. P. Frejaville, and E. Fournier. 1974. Intoxication by Hydrogen Sulfide. Eur. J. Toxicol. Environ. Hyg. 7(1):30-31 (Fre).

C-5. No exposure data. Does show nice gradation of effects. The same cases are described in Gaultier et al. (1967) [4-205].

4-250 Poda, G. A. 1966. Hydrogen Sulfide Can Be Handled Safely. Arch. Environ. Health 12:795-800.

B-4. A discussion of the occurrence (123 cases in 7 y) and the symptoms of H_2S in a heavy water plant which had an alarm system set for 10 ppm H_2S (it never went off). Contrary to other reports, little eye irritation was seen. Weakness, dizziness, nervousness, and nausea were the main effects. Work practices, treatment, and precautionary measures are also given.

4-305 Pohl, J. 1887. The [Toxicological] Action of Hydrogen Sulfide and Alkali Sulfides. Arch. Exp. Pathol. Pharmakol. 22:1-25 (Ger).

D--. Most animal experiments studied the toxicity of Na_2S. In two experiments, convulsions were induced by H_2S in rabbits by nasal insufflation or by introduction directly into the trachea.

4-091 Pollard, G. D., and D. W. Hirsch. 1978. In-Flight Toxicology of Fixed and Rotary Wing Aircraft Crew Stations. Paper No. 18, AGARD [Advisory Group for Aeronautical Research and Development] Conf. Proc. Vol. 225. 7 pp.

D--. After the firing of the guns, 2 air samples contained 63 or 126 ppm H_2S and 21 or 18 ppm HCN, as well as other gases.

4-313 Preuschen, G. 1974. Air Pollution and Human Work Capacity. In: Proceedings of the International Livestock Environment Symposium. University of Nebraska, Lincoln, Nebraska. April 17, 18, and 19, 1974. American Society of Agricultural Engineers. St. Joseph, Michigan. pp. 195-198.

D--. Workers exposed to animal house atmospheres experienced coughing, dizziness, shortness of breath (no numbers given) and decreased respiratory functions (no statistical evaluations done). No listing of contaminants or concentrations given.

4-177 Prouza, Z. 1970. Group Poisoning with Hydrogen Sulphide in an Unusual Situation on a Viscose Plant. Prakt. Lek. 50:27-29 (Czech); English translation available John Crerar Library, Chicago, Illinois. Order No. 5828.4F (22836).

B-5. Brief exposure to ~15-35 mg H_2S/m^3 caused minor symptoms in 4/9 workers. All 9 fully recovered. One man exposed to 4,000 mg/m^3 died very quickly.

4-276 Renne, R. A., and K. E. McDonald. 1979. Toxic Effects of Geothermal Effluents: Acute and Subacute Inhalation Toxicology of Hydrogen Sulfide and Ammonia in Rodents. In: Pac. Northwest Lab. Annu. Rep. 1979 DOE. Assist. Secr. Environ. Pt. 1: Biomedical Science. p. 275.

C--. Part of an annual report, just a summary provided. An initial 7-d exposure to 100 ppm H_2S (alone or in combination with 250 ppm NH_3) caused no significant changes in rats or guinea pigs. Subsequent 7-d exposure to 220 ppm H_2S caused a mild increase in the incidence of acute respiratory tract inflammatory lesions in guinea pigs. Subsequent 7-d exposure instead to 250 ppm NH_3 caused the same changes as well as mild interstitial pneumonitis and mild chronic nephritis.

4-337 Renne, R.A., and K.E. McDonald. 1980. Toxic Effects of Geothermal Effluents: Subacute Inhalation Toxicology of Hydrogen Sulfide and Ammonia in Rodents. In: Pac. Northwest Lab. Annu. Rep. 1980 DOE. Assist. Secr. Environ. Pt. 1: Biomedical Science. p. 240.

C--. Part of an annual report, so this is only a summary. Exposure for 22 d to 220 ppm H_2S, 250 ppm NH_3, or a combination of these gases had no effect on the major organs in rats and only slight respiratory and renal effects in guinea pigs.

4-274 Robinson, A. V. 1979. Toxic Effects of Geothermal Effluents: Effect of in Vitro Exposure to Hydrogen Sulfide on Cultured Rabbit Alveolar Macrophages. In: Pac. Northwest Lab. Annu. Rep. 1979 DOE. Assist. Secr. Environ. Pt. 1: Biomedical Science. pp. 279-281.

C-6. A summary of work in progress. Cultured rabbit alveolar macrophages were exposed to 50 or 200 ppm H_2S under various exposure regimes and times. Relative phagocytic ability and viability decreased with increasing concentration and time.

4-338 Robinson, A.V. 1980. Toxic Effects of Geothermal Effluents: Effect of in Vitro Exposure to Hydrogen Sulfide on Rabbit Alveolar Macrophages Cultured on Gas Permeable Membranes. In: Pac. Northwest Lab. Annu. Rep. 1980 DOE. Assist. Secr. Environ. Pt. 1: Biomedical Science. pp. 235-239.

C--. Exposure of rabbit alveolar macrophages to 60 ppm H_2S for 24 h caused a decrease to ~5% of the controls when cultured on gas-permeable membranes (more similar to in vivo exposure), and had no effect when cultured on gas-impermeable flasks. Cytotoxicity was more pronounced under continuous than intermittent exposure. Full expression of phagocytic ability depended on the length of time after the end of exposure as well as the duration and level of exposure.

4-275 Robinson, A. V., K. E. McDonald, and R. A. Renne. 1979. Toxic Effects of Geothermal Effluents: Effect of in Vitro Exposure of Hydrogen Sulfide on Free Cells Obtained from Lungs of Rats and Guinea Pigs. In: Pac. Northwest Lab. Annu. Rep. 1979 DOE. Assist. Secr. Environ. Pt. 1: Biomedical Science, pp. 282-283.

C-6. An abstract of work in progress. The numbers and types of lavaged lung cells from rats and guinea pigs exposed to 200 ppm H_2S for 24 h were indicative of an inflammatory response in the respiratory tract.

4-294 Robinson, L. F., M. N. Camp, and E. C. Chamberlain. 1942. A Source of Industrial Hazard from Hydrogen Sulfide Gas. Southern Med. J. 35(6):621-623.

D--. A short review of the effects of H_2S. And a case report of acute and subacute H_2S poisonings (no concentrations given) causing unconsciousness or eye inflammation.

4-272 Rosenberger, G., Ed. 1970. Sulfur. In: Diseases of Cattle. Paul Paley Verlag, Berlin and Hamburg, Federal Republic of Germany. pp. 1172-1174 (Ger).

D--. Atmospheric concentrations of H_2S >0.03 vol. % (300 ppm) are unhealthy for cattle. Concentrations of 0.1 vol. % H_2S are fatal within a short time. Symptoms and histopathology are described. The recommended maximum concentration to be allowed is 0.01 vol. %.

4-092 Rotenberg, Y. S. 1974. Correlation Between the Toxicity of Chemical Agents and Their Inhibitory Action on Isolated Mitochondria. Byull. Eksp. Biol. Med. 78(7):65-68 (Russ).

D--. A correlation was found between the LD_{50} and CL_{50} values for 29 compounds and their concentrations causing 50% inhibition of mitochondrial respiration. H_2S was included.

4-251 Rubin, H., and A. Arieff. 1945. Carbon Disulfide and Hydrogen Sulfide: Clinical Study of Chronic Low-Grade Exposures. J. Ind. Hyg. Toxicol. 27:123-129.

D-10. A study of 100 workers, confounded by exposure to both H_2S (1-5.5 ppm) and CS_2 (1.9-20.5 ppm); the average length of employment was not given. Subjective symptoms, such as fatigue, and objective neurological signs were diverse and vague, leading authors to believe that the adverse effects, if any, were minimal. No comparison to workers without this exposure.

4-288 Ryazanov, V. A. 1960. New Data on Maximum Allowable Concentrations of Pollutants in the Air in the U.S.S.R. In: Proceedings of the Diamond Jubilee International Clean Air Conference, London, 1959. Arnold Marsh, Ed. pp. 175-176.

D--. A brief review of the types of information used to set standards in the U.S.S.R., including a description of the "electrocortical conditioned reflex" frequently used. The MAC's for 30 compounds are given. The one-time MAC for H_2S was 0.03 mg/m^3, and the 24-h avg. was 0.01 mg/m^3.

4-093 Safiullina, S. I. 1978. Effect of Hydrogen Fluoride, Phosphine, and Hydrogen Sulfide on Rats. Tr. NII Kraev. Patol. No. 32:28-33 (Russ).

D--. Because workers involved in the flotation of phosphorite ores are subjected to the action of PH_3 with H_2S or HF, albino rats were exposed to 0.39 mg PH_3/m_3 and 1.21 mg HF/m^3 or 12.1 mg H_2S/m^3 for 4 mo. Since such combinations are unlikely from automobile exhausts, this paper was not examined further.

4-185 St. Hill, C. A. 1966. Occupation as a Cause of Sudden Death. Trans. Soc. Occup. Med. 16:6-9.

D-4. Case reports of fatalities. Two were probably caused by loss of consciousness due to H_2S, followed by inhalation of sludge.

4-224 Saitanov, A. O. 1969. Heart Disease in Severe Acute Hydrogen Sulfide Poisoning. Kardiologiya 9(8):92-95 (Russ).

C-6. Another case report, complicated by x-ray evidence of tuberculosis. A description of the symptoms and treatment of the female victim is given. Heart damage seen belonging to the class of toxic myocardiodystrophy. Slow recovery. No exposure information given.

4-180 Sandage, C. 1961. Tolerance Criteria for Continuous Inhalation Exposure to Toxic Material--I. Effects on Animals of 90-Day Exposure to Phenol, CCl_4, and a Mixture of Indole, Skatole, H_2S, and Methyl Mercaptan. (ASTIA AD 268 783). Armed Services Technical Information Agency, Arlington, Virginia. 33 pp.

D-10. The mixture containing 20 ppm H_2S caused varying occurrence and degrees of lung, liver, and kidney damage in the 3 different species (monkeys, rats, and mice). Weight loss and low grade, fluctuating anemia occurred in all species.

4-225 Sarin, M. I., N. V. Sarina, N. D. Topolianskii, L. G. Karavchenko, and V. J. Bugaev. 1972. Acute Hydrogen Sulfide Poisoning. Klin. Med. (Mosk.) 50(1):99-101 (Russ).

C-7. Case histories of 6 accidental poisonings, with extensive medical information. In 2 cases, exposure to ~1,300 mg H_2S/L caused death in minutes to an hour. The authors hypothesize that the symptoms were caused in significant measure by the "disorder of the function of the reticulohypothalamic system of the brain stem."

4-094 Sartorius, R., and W. Jaeschke. 1977. The [German] Air Pollution Control Standards for Hydrogen Sufide are Correct. Umwelt. No. 1:30-31 (Ger).

D--. Some atmospheric H_2S levels in Germany, ranging from 0.05 to 1.09 $\mu g/m^3$ (near an industrial region).

4-320 Savio, T., and H. D. Johnson. 1972. The Effect of Acute Exposure to H_2S on the Sympatho-Adrenal System of the Goat. Trans. Mo. Acad. Sci. 6:177-178.

C--. Abstract only. Exposure of female goats to 10, 50, and 100 ppm H_2S for 11 d caused increased urinary secretion of the catecholamine metabolite 3-methoxy-4-hydroxymandelic acid (a measure of sympatho-adrenal function). The change may be due to the H_2S or to anxiety.

4-253 Sayers, R. R., C. W. Mitchell, and W. P. Yant. 1923. Hydrogen Sulfide as an Industrial Poison. U.S. Bureau of Mines, Department of the Interior. Reports of Investigations, Serial No. 2491, U.S. Department of the Interior, Washington, D.C. 6 pp.

C-7. Review of the general properties and toxicology of H_2S. Limited description of animal experiments--all eventually lethal. Concluded that acute poisoning in humans occurs at 0.07-0.08% (700-800 ppm) H_2S and subacute poisoning at levels as low as 0.005% (50 ppm).

4-095 Schaffernicht, H., D. Suessenbach, and P. Wedekind. 1975. Hydrogen Sulfide and Noise Exposure in Industrial Animal Production. Z. Gesamte Hyg. Grenzgeb. 19(3):177-180 (Ger).

D--. Incidence and industrial hygiene data.

4-255 Scheler, W., and R. Kabisch. 1963. The Antagonistic Action on Acute H_2S Poisoning in the Mouse via Methemoglobin Formation. Acta Biol. Med. Ger. 11:194-199 (Ger).

C-11. The LC_{50} in control mice was 430 ppm H_2S compared to 1,380 ppm H_2S when the mice were pretreated with 108 mg $NaNO_2$/kg due to the protective action of methemoglobin formation induced by $NaNO_2$.

4-226 Schmitz, W. 1971. Appearance and Manifestation of a Disease with Psychomotor Symptoms Due to an Acute Manure Pit Gas Intoxication in a Boy Who Previously Had Sustained Brain Damage. Z. Gesamte Hyg. Grenzgeb. 17(8):628-632 (Ger).

D--. Confounded by the earlier trauma and the presence of other gases.

4-342 Schulze, B., R. Ullmann, and H. Kirsch. 1979. Problems of Air Pollution with Hydrogen Sulfide Emitted from Rivers. Z. Gesamte Hyg. Ihre Grenzgeb. 25(3):219-221 (Ger).

D--. Three instances of communal air pollution by H_2S from wastewaters are described: (1) no H_2S concentrations were measured, but the citizens suspected that H_2S was causing health problems (not specified). The air pollution discolored Pb-based house paints; (2) three people died; no H_2S measurements were made; and (3) H_2S measurements were made but are not reported here. A connection between H_2S and health problems could not be established.

4-227 Schwander, D. 1972. Toxic Pulmonary Edema After Inhalation of Hydrogen Sulfide. Successful Treatment by Continuous Positive Pressure Ventilation. Cah. Anesthesiol. 20(7): 785-792 (Fre).

C-6. Case report of exposure to fumes from liquid manure causing unconsciousness, irritation of the mucous membranes, neurological symptom, and pulmonary edema.

4-097 Seuren, P. 1979. A Case of Hydrogen Sulfide Poisoning and Stench Nuisance in a Broiler Stockbreeding Farm Caused by Storage Finished Mushroom Manure. Tijdschr. Diergeneeskd. 104(9):383-384 (Dut).

D--. Symptoms of workers are described and attributed to H_2S poisoning. Histological study of one patient described. No exposure levels given.

4-228 Shaparenko, B. A., V. M. Foderman, and N. S. Drevniak. 1972. Use of Antidote Hydrogen Sulfide Prophylactic Inhalations in Persons Exposed to Metallic Mercury Vapor. Zh. Ushn. Nos. Gorl. Bolezna. 32(4):1-3 (Russ).

D--. Four courses a year of antidote inhalations of solutions containing 30-60 mg H_2S/L were a useful prophylaxis for mercury poisoning.

4-098 Shaparenko, B. A., R. I. She'inin, O. A. Lastkov, V. M. Foderman, and N. S. Drevniak. 1973. Morphological Changes of the Nasal Mucous Membrane in Experimental Animals in Micromercurialism and the Effect of Hydrogen Sulfide-Carbonate Inhalations. Zh. Ushn. Nos. Bolezn. 33(4): 37-39 (Russ).

D--. Inhalation of steam from a solution containing CO_2 and 30 mg H_2S/L was a useful method for prophylaxis or treatment when rats had been exposed to 40 mg Hg/m^3 for 20 min/d for up to 5 mo.

4-332 Shtrum, I. Ya. 1938. The Combined Action of Carbon Monoxide and Hydrogen Sulfide. Fiziol. Zh. SSSR. 24:624- 629 (Russ).

D--. At lethal concentrations, a mixture of H_2S and CO required a total dosage of 130% that of either poison alone to cause death in mice. When the "time of reflex" in rabbits was used as the criterion, it required 160% of either poison's dose alone to elicit the same effect with a mixture of H_2S and CO. The lowest H_2S concentration tested was 300 mg/m^3.

4-182 Simson, R. E., and G. R. Simpson. 1971. Fatal Hydrogen Sulphide Poisoning Associated with Industrial Waste Exposure. Med. J. Aust. 1:331-334.

C-6. Post-mortem findings of the victim, exposed to H_2S levels possibly as high as 12,000 ppm.

4-099 Smith, R. P., and R. E. Gosselin. 1979. Hydrogen Sulfide Poisoning. J. Occup. Med. 21(2):93-97.

D--. A review of the use of antidotes and some discussion of the mechanisms of toxicity and the similarities to cyanide poisoning.

4-229 Sof'ina, L. I. 1969a. Effect of a Complex of Saturated and Unsaturated Hydrocarbons and Hydrogen Sulfide on the Activity of Certain Enzymes of the Brain, Liver, and Blood Serum. Gig. Tr. Prof. Zabol. 13(7):41-43 (Russ).

D--. Albino rats were exposed to cracking gases 6 h/d for 4 mo. The cracking gas in the exposure chamber contained 100 mg H_2S/m^3. The effects of H_2S on the enzymes would be confounded by those of the saturated and unsaturated hydrocarbons in the cracking gas.

4-230 Sof'ina, L. I. 1969b. Effect of Sulfur Petroleum Reprocessing Gaseous Products on the Activity of Some Rat Brain Enzymes. Tr. Ufim. Nauch.-Issled. Inst. Gig. Prof. Zabol. 5:64-68 (Russ).

D--. Exposing rats to 1,200 mg H_2S/m^3 for 2 h/d for 5 d lowered the activities of glutaminase (by 34.5%), alkaline phosphatase (by 29%), and malate dehydrogenase. Sorbitol dehydrogenase activity was increased. (Cracking gas caused the most expressed changes in brain enzyme activities.)

4-183 Sorokin, G. E., and N. M. Olshanskaya. 1941. Electrocardiographic Studies on Changes in Cardiac Activity Induced by Hydrogen Sulfide. Fiziol. Zh. 30:530-532 (Russ).

D--. Solutions containing 265 mg H_2S/L were injected intravenously into rabbits.

4-184 Spolyar, L. W. 1951. Three Men Overcome by Hydrogen Sulfide in Starch Plant. Ind. Health Monthly 11:116-117.

C-6. H_2S levels of 300-400 ppm caused unconsciousness, and 1 death.

4-101 Spurgeon, J. C. 1978. The Correlation of Animal Response Data With the Yields of Selected Thermal Decomposition Products for Typical Aircraft Interior Materials. NTIS No. AD-A062938, National Technical Information Service, U.S. Department of Commerce, Springfield, Virginia. 40 pp.

C-12. The times to incapacitation and death of at least 9 rats exposed to mixed combustion products, the gas containing 0-14.2 mg H_2S/g sample.

4-102 Stine, R. J., B. Slosberg, and B. E. Beacham. 1976. Hydrogen Sulfide Intoxication: A Case Report and Discussion of Treatment. Ann. Intern. Med. 85(6):756-758.

D-4. A case report of cyanide-type treatment for H_2S poisoning.

4-103 Stolpe, J., R. Sedlag, and B. Bresk. 1976. Single and Complex Effect of Ammonia and Hydrogen Sulfide in the Air on Small Laboratory Animals (Rats) Under Various Environmental Conditions. 2. Effect of Hydrogen Sulfide Alone and Hydrogen Sulfide with Ammonia and Dust. Arch. Exp. Veterinaermed. 30(4):541-545 (Ger).

B-10. Rats exposed to 10 ppm H_2S, 10 ppm H_2S and 50 ppm NH_3, 5 ppm H_2S and 30 ppm NH_3, or 5 ppm H_2S and 30 ppm NH_2 and dust caused varying decreases in weight gain and food utilization. All test groups were at 10°C. Cold controls and optimal temperature controls were done.

4-104 Strack, W. 1977. Danger from Hydrogen Sulfide Used in Modern Technologies for Leather Production. Koza Obuca. 26(11):283-285 (Croat).

D--. The occurrence of an accidental death from H_2S used in the drum bating and pickling process (short liquor) is described.

4-186 Sukhanova, V. A. 1962. The Influence of the Products of Processing High-Sulfur Crude Oil on the Functional State of the Gastrointestinal Tract (Experimental Study). Gig. Tr. Prof. Zabol. 6:7-11 (Russ).

D--. Rats and dogs were exposed to atmospheres containing cracking gases with 75-77 vol. % saturated hydrocarbons, 13-15 vol. % unsaturated hydrocarbons, 4-7 vol. % hydrogen, and 3-6 vol. % H_2S. Four dogs exposed 4 h/d for 5 mo in a chamber containing 50-70 mg H_2S/m^3 and the other components did not show any change in blood amylase. Rats exposed similarly for 4 mo showed a drop in uropepsin level in the urine and reduced intensity of incorporation into the pancreas proteins of radiolabeled methionine.

4-289 Taiganides, E., and R. K. White. 1969. The Menace of Noxious Gases in Animal Units. Trans. Am. Soc. Agric. Eng. 12(3): 359-362.

D--. A review of the occurrence of H_2S, NH_3, CO_2, and CH_4 in animal units, and their toxicities to different species. Includes a discussion of potentially lethal situations and preventive measures.

4-327 Teleky, L. 1931. Industrial Hygienic Studies on Hydrogen Sulfide Poisoning. Dtsch. Med. Wochenschr. 57:1026-1027 (Ger).

C--. Case histories of H_2S poisoning are described. Among them is the chronic poisoning of a family by H_2S (no concentrations given) emanating from a wall constructed of porous bricks in their apartment. On the other side of the wall was an accumulator (storage battery) room. The sulfuric acid vapors apparently reacted with the construction materials to form H_2S.

4-105 Thiele, V. 1979. Experimental Studies on Determining an Odor Threshold Value for Hydrogen Sulfide. Staub-Reinhalt. Luft. 39(5):159-160 (Ger).

C-9. The odor threshold value was 0.0016 mg/m^3, the 50-percentile value of a dose-response curve from 2 groups. The responses for "annoyance" values were more widely distributed, the 50% value ranging from ~0.002 to ~0.030 mg/m^3.

4-231 Thiess, A. M. 1968. Case Contribution of Hydrogen Sulfide Poisoning with Fatal Outcome. Zentralbl. Arbeitsmed. 18(12):366-368 (Ger).

C-7. Short exposure to unknown levels of H_2S caused only slight symptoms of H_2S intoxication: headache, vomiting, and light irritation of mucous membranes and conjunctiva of the eye. Death was due to a skull fracture, not H_2S poisoning.

4-187 Thoman, M. 1969. Sewer Gas--Hydrogen Sulfide Intoxication. Clin. Toxicol. 2:383-386.

D-5. The accidental exposure of 5 people to H_2S fumes, symptoms ranging from mild discomfort to near fatal. Concentrations and exposure times unknown.

4-106 Timofeev, V. P. 1974. Biological Effect of Natural Human Metabolites in a Dwelling Environment. Tr. Leningr. Sanit.-Gig. Med. Inst. 105:168-171 (Russ).

D--. White male rats (18 per group) were exposed to a five-component mixture of human metabolites for 100 d. In the eight tests, the average concentrations of the components were 0.92 to 74.4 mg CO/m^3, 0.20 to 148.3 mg NH_3/m^3, 0 to 8.75 mg H_2S/m^3, 0.3 to 240.1 mg acetone per cubic meter, and 0.009 to 5.6 mg phenol per cubic meter. H_2S was judged from partial coefficients of multiple linear regression as contributing 16% to the animals' deaths.

4-107 Tomaszewska, Z. 1978. Multiple Fatal Hydrogen Sulfide Poisonings. Arch. Med. Sadowej Kryminol. 28(1):55-57 (Pol).

C-8. Four men exposed to ≦944 mg H_2S/m^3 were fatally poisoned while cleaning a settler for sewage from a carriage washer.

4-108 Tonzetich, J., and S. K. Ng. 1976. Reduction of Malodor by Oral Cleansing Procedures. Oral Surg., Oral Med. Oral Pathol. 42(2): 172-181.

B-9. A group of 8 people found the threshold of objectionability (not odor detection) for H_2S to be 0.15 mg/m^3. This was a small part of a larger study.

4-188 United States of America Standards Institute. 1967. Acceptable Concentrations of Hydrogen Sulfide, USAS Z37.2-1966, USASI, New York, New York. 8 pp.

C--. Summary of properties and toxic effects of H_2S, similar to other reports. Has recommended acceptable concentrations: ten-min-peak exposure 50 ppm; 8-h 5 d/wk, 20 ppm; and level to avoid any discomfort, 10 ppm. More recent issues of this H_2S standard are available.

4-189 United States of America Standards Institute. 1972. Acceptable Concentrations of Hydrogen Sulfide. USAS Z37.2-1972, USASI, New York, New York. 8 pp.

A--. A review of H_2S occurrence, properties (physical, chemical, toxic), and detection methods. At low concentrations the predominant effects are on the eyes (≧10 ppm) and respiratory tract (≧50 ppm). No permanent after-effects in cases of recovery unless there was prolonged O_2 depletion. No evidence of accumulative or systemic effects following repeated exposures.

5-424 Urban, C. M., and R. J. Garbe. 1980. Exhaust Emissions from Malfunctioning Three-Way Catalyst-equipped Automobiles. S.A.E. Tech. Pap. Ser. 800511. 11 pp.

C--. Maximum emission rates (mg/m^3) in 4 malfunctioning, 3-way catalyst-equipped automobiles:

NH_3	254
CN	67
H_2S	8
HCHO	3

4-174 U.S. Geological Survey Outer Continental Shelf. 1976. Outer Continental Shelf Standard--Safety Requirements for Drilling Operations in a Hydrogen Sulfide Environment. Report No. GSC-OCS-1. Geological Survey Conservation Division, U.S. Department of the Interior, Reston, Virginia. 15 pp.

D--. An outline of safety procedures and equipment to be used. Protective breathing apparatus to be worn when H_2S levels reach 20 ppm, and nonessential personnel evacuated at 50 ppm.

4-173 U.S.P.H.S. United States Public Health Service. 1964. The Air Pollution Situation in Terre Haute, Indiana with Special Reference to the Hydrogen Sulfide Incident of May-June, 1964. PB 227 486. National Technical Information Service, U.S. Department of Commerce, Springfield, Va. 28 pp.

A-6. Limited by the lack of tests for non-sulfide contaminants. Mild (non-hospitalization) complaints of odors and symptoms at 20-30 ppb H_2S. Recommends an ambient air quality level of 50 ppb/1 h.

4-337 U.S.S.R. State Committee of the Council of Ministers for Construction. 1972. Sanitary Norms for Industrial Enterprise Design. Publishing House of Literature on Construction, Moscow. 92 pp. (Russ).

C--. In the USSR, the MAC for H_2S in workplaces was 10 mg/m^3, and in populated places was 0.008 mg/m^3 (one-time and avg.).

5-413 Van Gemert, L. J., and A. H. Nettenbreijer. 1977. Compilation of Odour Threshold Values in Air and Water. National Institute for Water Supply. Leidschendam, the Netherlands, and Central Institute for Nutrition and Food Research, TNO, Zeist, the Netherlands.

A--. Compilation of odor threshold values reported by different researchers, for many compounds, including:

Ammonia	0.03-37 mg/m^3
HCN	< 1.1-6
H_2S	0.001-2
HCHO	0.033-2.2
Methanol	4.3-11,700
Acrolein	0.05-4.1

4-110 Vasil'eva, I. A. 1973. Effect of Small Concentrations of Carbon Disulfide and Hydrogen Sulfide on the Menstrual Function of Women and the Estrous Cycle of Experimental Animals. Gig. Sanit. 38(7):24-27 (Russ).

D--. Interaction study. Workers were exposed to ≤ 5 mg H_2S/m^3 in 71.1% of the samples and no sample exceeded the MAC. They were also exposed to CS_2 in concentrations up to 12.6 mg/m^3. They showed a higher incidence of ovarian function disturbances. The estrous cycle of rats similarly exposed was prolonged.

4-1111 Vasil'eva, I. A. 1975. State of Reproductive Functions in Chemical Industry Workers. Gig. Tr. 11:179-182 (Russ).

C-4. During pregnancy, an unknown number of women workers in a viscose spinning plant exposed to unknown concentrations of H_2S and CS_2 experienced incidences of 15.0 and 16.8% toxicosis of early and late pregnancy compared to 7.8 and 8.6% in rewinding-sorting plant workers and 10.7 and 2.9% in housewives. The spinning plant workers experienced significantly ($p<0.001$) more blood loss during birth, and their offspring showed twice as many developmental flaws and malformations than those of the housewives. The spinning plant workers had chromosomal aberrations.

4-261 VDI, Verein Deutscher Ingenieure [Society of German Engineers] Committee on Air Purification. 1970. Permissible Immisson Concentrations of Hydrogen Sulfide. PB-221 243-T, National Technical Information Service, U.S. Department of Commerce, Springfield, Virginia. 12 pp.

D--. Review of the occurrence and properties of H_2S, and its effects on man, animals, and plants. The exposure limits given are: ~0.15 mg/m^3 (as half-hour mean value) for the Permissible Immission Concentration (PIC) for continuous exposure; ~0.3 mg/m^3 (3 x a day half-hour mean value) for the PIC for intermittent exposure; and ~30 mg/m^3 for the Permissible Work-Station Concentration.

4-308 Vigil, P. J. 1979. A State-of-the-Art Review of the Behavioral Toxicology of Hydrogen Sulfide. UCRL-15093. Reference Dynamics, Santa Rosa, California. 55 pp.

B--. Review of studies of acute, subacute, chronic, and low level H_2S exposure of humans. It concludes that behavior toxicology as a minimum toxic effect is likely, but not proven, and no threshold is definable due to lack of good low-dose data.

4-113 Vilisov, B. A., L. M. Kremko, Z. N. Pavlyutina, and V. M. Ermakova. 1975. Working Conditions and Illness Rate of Workers in the Biological Flax-Retting Division. Gig. Tr. Prof. Zabol. No. 6:43-45 (Russ).

D--. The above named workers were exposed not only to H_2S but also to NH_3, CO_2, aliphatic amines, monocarboxylic acids, bacteria, and molds.

4-260 von Rad [no initial], and Scheidemantel. 1933. Late Consequences of a Hydrogen Sulfide Intoxication (Perturbation of the Heart Activity and Lesions of the Central Ganglia). München. Med. Wochenschr. 80:1494-1495 (Ger).

D-4. Description of an accidental exposure, where 0.5 h after the accident there was 0.83 mg (volume not given) H_2S. For several months, the victim complained of insomnia, and later had an epileptic attack. Victim still suffered from high blood pressure and abnormal irritability of the vasomotoric center 1.5 y after the accident.

4-114 Wachnik, Z. 1974. Gases Harmful to Birds. Med. Weter. 39(10): 605-608 (Pol).

D--. A Polish review of bird intoxication by NH_3, H_2S, and CO.

4-190 Wakatsuki, T. 1959. Experimental Study on the Poisoning by Carbon Disulfide and Hydrogen Sulfide. Shikoku Igaku Zasshi. 15:671-700 (Japan).

C-8. Rated only from English abstract. Rabbits exposed to 100 ppm H_2S 30 min/d for 4 mo showed only slight changes in peripheral blood picture, returning to normal shortly after exposure stopped. "General condition," body weight, and serum calcium and protein were unaffected. No direct respiratory information.

4-191 Walton, D.C., and M. G. Witherspoon. 1926. Skin Absorption of Certain Gases. J. Pharmacol. Exp. Therap. 26:315-324.

C-6. No inhalation exposure. Pure H_2S caused swelling and blackening when contacting small areas of guinea pigs skin, and death in 45 min when half of the guinea pig was exposed.

4-256 Weedon, F. R., A. Hartzell, and C. Stetterstrom. 1940. Toxicity of Ammonia, Chlorine, Hydrogen Cyanide, Hydrogen Sulphide, and Sulphur Dioxide Gases. V. Animals. Contrib. Boyce Thompson Inst. 11:365-385.

B-8. 8 rats and 4 mice were exposed to 16, 63, 250 or 1,000 ppm H_2S for a maximum of 23 h. There were no symptoms at 16 ppm for 16 h, and increasing morbidity and mortality for the other tests with increasing concentrations and exposure times.

4-328 Weise, W. 1933. Gastrointestinal Diseases from Chronic Inhalation of Carbon Disulfide and Hydrogen Sulfide. Arch. Gewerbepathol. Gewerbehyg. 4:219-279 (Ger).

B-9. Repeated exposure of very small numbers of rats and rabbits to 10, 100, or 1,000 ppm H_2S caused noticeable disturbance or changes in the gastrointestinal tract only at the higher levels.

4-232 Westbye, O. 1971. Composition and Toxicology of Manure Gas. Nor. Vet. Tidsskr. 83(10):522-525 (Nor).

D--. Review. Dose-response generalizations are tabulated for ammonia and hydrogen sulfide. The hygienic limit is given as 10 ppm for H_2S.

4-116 Westermann, H. D., A. Thalmann, and H. Kummer. 1975. The Toxicity of Hydrogen Sulfide in Animal Feeding: A Survey of the Literature. Landwirtsch. Forsch. 28(1):70-80 (Ger).

D--. A review with 42 references. Not pertinent to this study.

4-117 Williams, F. D., J. F. Emele, and M. C. Alford. 1977. The Application of the Dynamic Triangle Olfactometer to the Evaluation of Oral Odor. Chem. Senses Flavour. 2(4):497-502.

A-13. The odor threshold for H_2S was 0.27 mg/m^3 (0.19 ppm). Part of a larger study.

4-257 Winek, C. L., W. D. Collom, and C. H. Wecht. 1968. Death from Hydrogen-Sulphide Fumes. Lancet 1:1096.

D-5. Case report of a fatality after short exposure to 1,900-6,100 ppm H_2S. Autopsy revealed extensive respiratory pathology, and congested spleen and liver.

4-118 Winneke, G., J. Kotalik, H. O. Keldenich, and J. Kastka. 1979. Perception of Hydrogen Sulfide in Laboratory- and Field-Conditions. Staub-Reinhalt. Luft 39(5):156-159 (Ger).

C--. Using a group of 31 people, a detection limit for H_2S of 2.65 $\mu g/m^3$ was determined.

4-192 Yant, W. P. 1930. Hydrogen Sulphide in Industry--Occurrence, Effects, and Treatment. Am. J. Public Health 20:598-608.

B--. Review of toxicology of H_2S, both acute and subacute, and the occurrence and prevention of poisoning in the petroleum industry. Indicates a physiological threshold <50 ppm and an odor threshold of 0.13 ppm.

4-193 Yant, W. P., and H. C. Fowler. 1926. Hydrogen Sulphide Poisoning in the Texas Panhandle, Big Lake, Texas, and McCamey, Texas Oilfields. Bull. No. 2776. Bureau of Mines, U.S. Dept. of Commerce. 20 pp.

D--. H_2S concentrations in several different locations ranged from ~100 ppm to 100,000 ppm. Workers exposed to the lower concentrations had no symptoms while those at the higher levels had symptoms ranging from eye irritation and nausea to unconsciousness and death (at 1,000 ppm). Many dead rabbits and birds were found around one field. A long discussion on safety practices and equipment and treatment is included.

4-119 Zaitseva, D. M. 1976. Immunobiological Reactivity of the Bodies of Workers Producing the Sulfonate Additive PMS. Sb. Nauchn. Tr. Bashk. Gos. Med. Inst. 23(1):111-112 (Russ).

D--. Exposure of the title workers to SO_2, SO_3, NH_3, H_2S, hydrocarbons, acetic acid, and excessive noise and microclimatic conditions caused high morbidity due to acute respiratory diseases.

4-194 Zander, R. 1950. Carbonization Gas Intoxications. Dtsch. Gesundheitswes. 5:1422-1444 (Ger.).

C-6. Carbonization gas contains an avg. 2.0 vol % H_2S. This is confounded with the presence of other gases, particularly carbon monoxide (avg. 10.3 vol %). No patient exposure data are given. Several case reports given, no fatalities. Some EKG changes were noted in the patients.

4-126 Zhigunov, N. F. 1975. Techniques for Studying Industrial Environmental Factors on the Heterogeneity of O- and Vi Antibodies. Gig. Sanit. No. 11:767 (Russ).

D--. Effects due to H_2S are confounded by the higher concentrations of CS_2.

4-329 Ziaber, Z. 1978. Effect of Sulfide-Hydrogen Sulfide Bath on Sulfohydryl Groups (SH) in the Serum of Patients with Rheumatoid Arthritis. Pol. Tyg. Lek. 33(29):1147-1150 (Pol).

D--. Taking S-H_2S for ≧90 min significantly increased the SH groups in the serum in patients with rheumatoid arthritis. No information was given on the concentration of H_2S the patients might have inhaled or been exposed to by skin absorption.